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NEW SERIES, VOLUME XVIII

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HEAD INJURIES

AND SOME OF THEIR COMPLICATIONS*

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BOSTON, MASS.

THE post-bellum literature upon the subject of head injuries and their complications has been so voluminous and so comprehensive that a paper confined to the general classification and treatment of such injuries would under these circumstances seem hardly warranted. In two recent papers, Mock[1] and Coleman[2] have so wisely and sanely outlined the general management of these cases that their articles should be consulted by those desiring to inform themselves upon this aspect of the subject. What I have had in mind for consideration in this paper, therefore, is a brief picture of the various groups into which skull fractures naturally fall, together with a more extended and detailed story of certain individual cases, their treatment, complications and end-results so far as possible.

Patients who have suffered violence to their heads in one form or another may, as a rule, be put into one of the following categories:

Class I. Those who are conscious and rational, in whom the cerebral damage is probably slight, a clinical state which is usually spoken of as "concussion." This condition may or may not be associated with a skull fracture, and there is usually a brief period of loss of consciousness at the time of the accident. Such patients almost without exception do well if confined to bed for a period of one to two weeks, but of course should be kept under careful observation during the first forty-eight hours especially, in case pressure symptoms of a serious nature ensue. If headache or vomiting is troublesome magnesium sulphate by rectum is indicated or rarely one or more lumbar punctures. If a fracture is shown in the *x*-ray and it is linear, it may be disregarded entirely except as an indication to keep the patient relatively quiet for a somewhat longer period than when there has not been a fracture.

Class II. Those who show evidence of considerably more intracranial damage than is ordinarily implied by the term concussion, the head injury as a rule being associated with a demonstrable linear fracture of either the base or the vault. These are the patients who require extremely careful observation, the whole picture of pulse, respiration, blood pressure, spinal pressure and especially the state of consciousness being taken into account in deciding whether they should have merely dehydration, or dehydration plus lumbar punctures, or in occasional instances a decompression.

In the following case there was evidence of contre-coup cerebral damage but the

* From the surgical service of the Peter Bent Brigham Hospital, Boston.
Read before The Associated Physicians of Montclair, N. J., Nov. 20, 1931.

treatment was non-operative and the subsequent story seems to have borne out the wisdom of this method.



FIG. 1. Case II, at time of discharge to illustrate linear scar of subtemporal decompression

CASE I. L. M. A., a boy of six, surgical No. 28583, was referred to the Peter Bent Brigham Hospital by Dr. S. A. Levine of Boston, on April 11, 1927.

Five or six hours before admission the child had been in a motor accident and was rendered unconscious. There were multiple lacerations of the scalp and face, the right pupil was larger than the left and there was apparently some weakness of the right arm and leg. There was likewise a bilaterally positive Babinski reaction. *X*-rays of the skull showed a linear fracture of the right frontal bone, close to the coronal suture, crossing the middle meningeal channel in the floor of the middle fossa.

The child was put under careful observation after his scalp and face lacerations were sutured. He showed no signs of shock, but remained unconscious. Lumbar puncture was performed and yielded 20 c.c. of pink, bloody fluid.

He remained practically unconscious for four days but would take nourishment during this time. His right face showed some weakness



FIG. 2. Case III, showing appearance at time of discharge.

as well as the right arm and leg as noted, evidently a contre-coup contusion as his fracture was on the right side.

He began to talk ten days after the injury, his right-sided weakness gradually cleared up, so that at the time of his discharge on May 2, 1927, he seemed quite normal in every way, including speech comprehension.

End-result (two years): His mother reported on May 6, 1929, that he had remained in excellent physical and mental condition and that he stood well in school although the youngest in his class.

If pressure symptoms persist and become serious a decompression occasionally may be advisable. The next case well exemplifies such a situation.

CASE II. J. D., a boy fifteen years of age, surgical no. 25406, was referred by Dr. A. C. Freeman of Norwich, Conn., on December 16, 1925.

Two days before admission he had been knocked down by a motor and rendered unconscious. He was taken to the local hospital



FIG. 3. Case IV. *X*-ray showing crescentic fracture lines in left frontal region (preoperative).



FIG. 4. Case IV. Operative scar at time of discharge.



FIG. 5. Case V. *X*-ray showing lateral view of depressed fracture in left temporoparietal region (preoperative).



FIG. 6. Case V. Appearance of patient and of operative site at time of discharge.

where he began gradually to regain consciousness. I saw the boy at the Norwich Hospital on the night of December 15. He

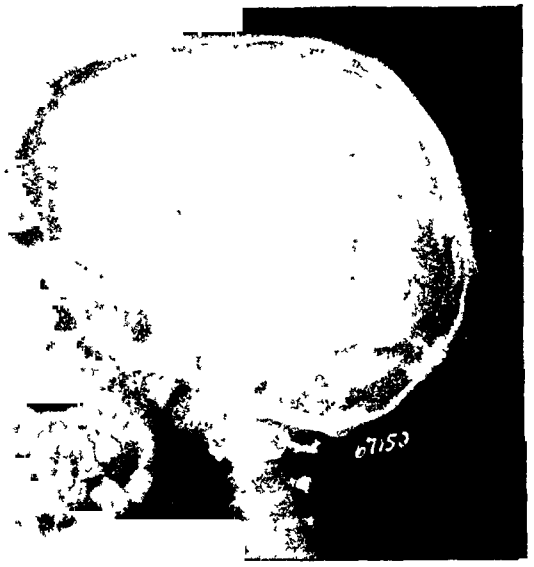


Fig. 7. Case VI. *X*-ray illustrating huge depressed and fragmented area in left temporo-occipital region (preoperative).

was extremely drowsy but would respond to simple requests. There was a boggy swelling over the occipital region. *X*-rays of the skull showed a fracture with some depression at this site and a linear fracture extending forward to the right temporal region.

First Operation: 3 A.M., December 16. The depressed area was explored and excised. The dura was contused but not penetrated. Some bloody fluid was evacuated by a nick in the dura and 10 c.c. of clear fluid removed by a puncture of the ventricle. The child was given magnesium sulphate solution by rectum, but he remained drowsy and was therefore brought up to Boston on the evening of December 16. The next day there was apparent some weakness of the left face, arm and leg, and he remained extremely drowsy. It was thought that he might have a clot on the right side.

Second Operation: December 17. A right subtemporal decompression was performed through the usual linear incision in the right temporal region. A large amount of bloody subdural fluid was evacuated. The cortex was considerably contused but no clot was present. The dura was opened widely over the temporal lobe and the wound closed without drainage.

On December 20 the boy had a series of convulsions involving the left side of his body. This was the only such episode. On

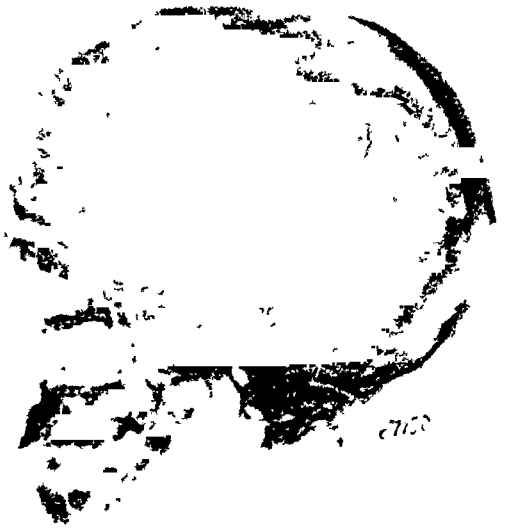


Fig. 8. Case VI. Appearance of fracture area after operation. Two of largest fragments have been replaced and depressed lower edge re-elevated. Small white lines are silver clips used in hemostasis.

December 26 he was quite conscious and rational, with the strength of his left side much improved. He was discharged January 6, 1925, apparently quite well in every way. (Fig. 1.)

End-result (two years): February 10, 1928. He went back to school a few weeks after his return home in January 1925, and got along very well. In the autumn of 1927 he entered college and has been entirely free of any symptoms. He has never had convulsions since the single series in the hospital.

Class III. Those who are profoundly unconscious or even moribund when they come under observation. Such patients have stertorous breathing, either a feeble or markedly bounding pressure pulse according to how far shock is advanced, sweating, restlessness, and all evidences of fatal cerebral damage. The face and scalp are as a rule highly contused and lacerated, the eyelids closed with ecchymotic edema and there is frequently bleeding from one or all cranial orifices. X-rays of the skull are likely to show a so-called bursting fracture or fractures, and

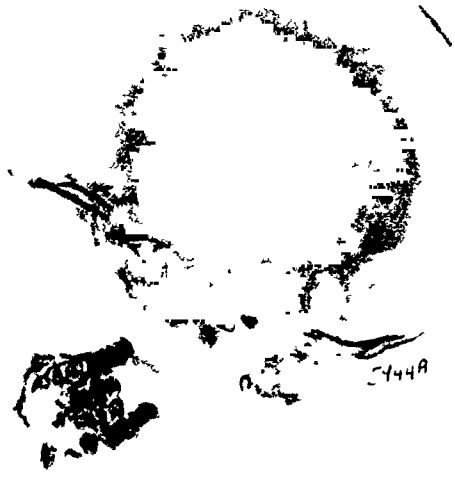


FIG. 9. Case VII. *X*-ray showing appearance of fragmented area before operation with area of bone defect in its center.



FIG. 10. Case VIII. *X*-ray before operation showing long linear fracture with crescentic depressed fracture at its posterior end.



FIG. 11. Case VIII. Appearance of patient and scar at time of discharge.



FIG. 12. Case IX. Preoperative x-ray showing fragmented bullet and loose indriven bone pieces around small wound of entrance.

lumbar puncture if it is performed yields fluid which is almost wholly blood. Little if anything can be done for these patients although the usual measures for combatting shock should be instituted with perhaps occasional small doses of morphia. In years past we have been led rarely into performing a decompression in the faint hope of helping such a condition, but I do not recall a single instance of survival. It is my feeling that an operation is useless, even harmful in this type of case.

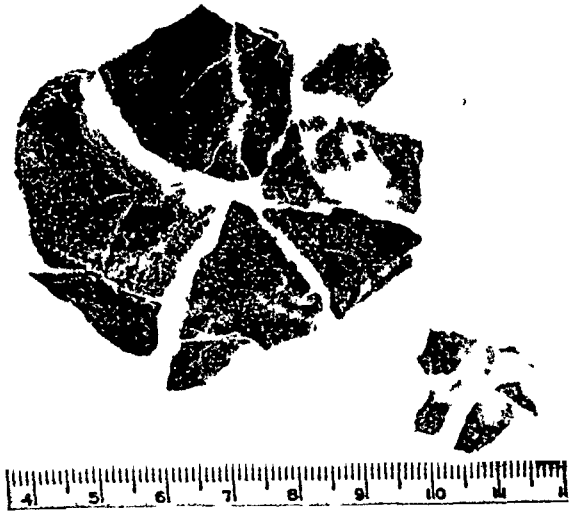


Fig 13. Case x Bone fragments removed.

Class iv. Those who, either from their history or while under observation present the typical syndrome of an extradural clot from meningeal hemorrhage. In these patients the commonest story is that they have been rendered unconscious for a brief period and after regaining consciousness have relapsed gradually into a state of great drowsiness or actual coma. The interval of lucidity may vary from an hour or two to a couple of days, though rarely longer, and I have known a patient to have several episodes of alternating consciousness and coma over the course of a week. Skull plates almost invariably show a linear fracture crossing the meningeal channel in the temporal fossa. As soon as the condition is recognized a subtemporal decompression should be performed by a linear incision through the temporal muscle. The clot is often an inch in thickness and may be spooned or gently sucked off from the dura. By this means the torn meningeal artery is disclosed and ligated either by a stitch or by a silver clip. The muscle, galea and skin are then closed, leaving a small rubber tissue drain for twenty-four hours at the lower end of the wound. By the end of the operation, which should be performed under local anesthesia, the patient in most instances has once more become conscious.



Fig. 14. Case x. Pinch grafts on granulating area.

The following case is a fairly typical instance of this class of injury.

Case iii. S. F. Z., a six-year-old boy, surgical no. 26509, was referred by Dr. M. A. Gilbert of Chelsea, Mass. on June 8, 1926.

He had fallen off the end of a truck twenty-four hours before admission, was picked up unconscious and taken to his home. He regained consciousness after fifteen minutes, cried and complained of pain in his head. He continued to have pain in his head throughout the night and until noon the next day, when he lapsed into coma and was brought to the hospital.

Examination showed the boy to be extremely restless and thrashing about the bed. There was an obvious left facial weakness and apparently some lack of use of the left arm and leg, with a positive Babinski reaction on the left. Abdominal reflexes were absent on the left, and the right pupil was larger than the left. The *x*-ray showed a linear crack in the right temporal region.

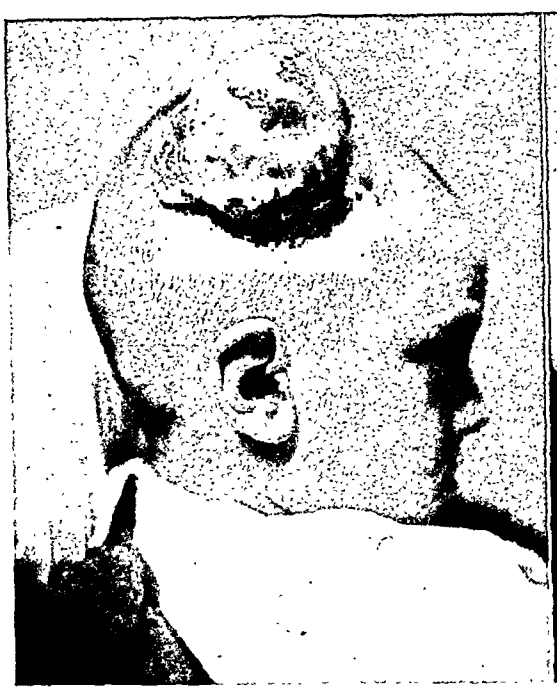


Fig. 15. Case xi. Huge fungus cerebri present at time of admission.



Fig. 16. Case xi. Fungus completely receded at time of patient's first discharge from hospital.

Operation: A right subtemporal decompression was performed one hour after admission. An extradural clot averaging 2 cm. in thickness and 10 cm. in diameter was removed. The middle meningeal artery was found to be torn in two of its branches. These were ligated with silver clips. A small rubber tissue drain was left beneath the dura.

The patient was conscious and rational the next morning. He made an uneventful convalescence and at the time of his discharge (June 25, 1926), was perfectly well. (Fig. 2.)

End-result (one year): He reported on July 30, 1927, at which time he was apparently in the best of health. His decompression area was completely ossified.

There are exceptions to all rules, and since the case just described is almost of the textbook variety it may be well to give the details of one which was not so clear.



Fig. 17. Case xii. Appearance of patient and operative flap at time of discharge.

Case iv. L. R., a girl of sixteen, surgical No. 25047, was referred on October 23, 1925, by Dr. C. Frothingham of Boston.

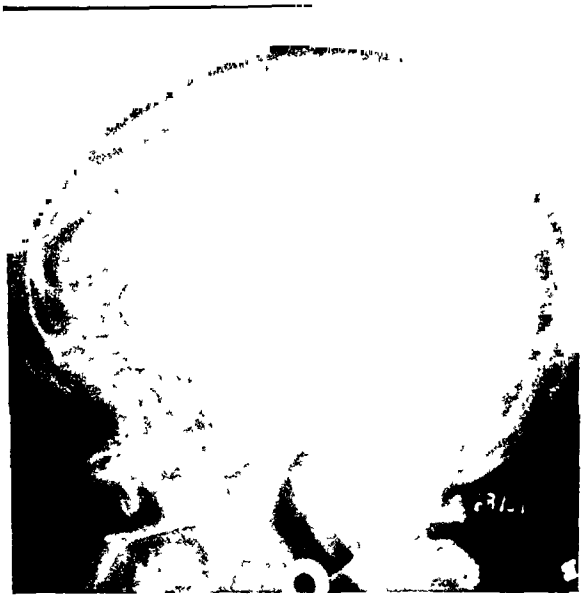


Fig. 18. Case xiii. Preoperative *x*-ray at time of admission showing huge fragmented area in frontal region extending into frontal sinus.



Fig. 19. Case xiii. *X*-ray showing lateral view of large aerocele within right frontal lobe.

She had been thrown from a horse a few hours before admission, but not rendered unconscious. She got up, walked over to some friends and was taken home. She became somewhat drowsy after getting home and was therefore brought into the hospital, where she continued to be drowsy. She had abrasions over the face and some swelling of the left frontal region. *X*-rays showed a stellate, very slightly depressed fracture of the left frontal bone. (Fig. 3.) This depression would not of itself have been a sufficient indication for operation. The patient was kept under careful observation for forty-eight hours. She became somewhat more drowsy and the edges of the optic discs showed some blurring in spite of magnesium sulphate by rectum. Her left pupil became smaller than the right, and her pulse fell to 66 at times. Taking all facts into consideration, an exploration of her fractured area seemed advisable.

Operation: October 25, 1925. A small osteoplastic flap including the fractured area was turned down. There was apparent immediately an extensive extradural clot 2.5 cm. in thickness, covering the exposed field, and extending down over the tip of the frontal lobe. This was removed by the suction apparatus, and bleeding from small meningeal branches controlled by bits of muscle. The bone flap was then replaced, no attempt being made to do anything to the fractured fragments as there was practically no depression. It may be said that the dura was opened slightly and the cortex found to be normal in appearance. A small rubber tissue drain was left.

She made an uneventful recovery and was quite free of any symptoms at the time of her discharge, November 10, 1925, at which time her pupils were again equal. (Fig. 4.)

End-result (two years): On November 17, 1927, she reported that she was perfectly well.

Class v. *Depressed Fractures.* These, of course, may be of any size and variety, and may involve any area of the cranial vault. They may be seen immediately after the accident or may be brought in for treatment many days or weeks after injury. Neurological signs and symptoms are often present, varying according to the region of cortex that has been damaged.

The treatment is practically always operative unless the depression is extremely slight and there are signs of neither cortical irritation nor compression. The optimum

procedure varies of course with the conditions found. In some cases it may be possible to make a burr opening just outside the depressed bone and by sliding a smooth instrument between bone and dura to elevate the fracture into its normal position. In most cases the whole depressed area will have to be excised, usually best by several burr holes being made around it and by these being connected with cutting forceps. The whole block of bone can then be lifted out, thus ample room being afforded to deal with any hemorrhage. This block should be preserved in sterile gauze until just before closure of the wound, when it can often be flattened out and replaced upon the dura, or if this is not possible the individual pieces may be detached and placed in the same way so that they may form the framework for covering over a large portion of the defect.

A few examples giving some unusual features may be cited:

Case v. I. C., a girl nineteen years of age, surgical No. 28010, was brought into the hospital through the accident room on January 16, 1927.

The meager history stated that she had been in a motor accident less than an hour before admission, and had been thrown forward against the windshield. She had been rendered unconscious momentarily.

Examination showed a star-shaped wound behind the right ear with brain tissue extruding. There was hypesthesia of her whole left side. She was conscious and rational, but highly excited at the time of entrance. *X*-rays showed a depressed fracture in the right temporoparietal region with a large quadrilateral fragment 3 × 3 cm. in diameter and a smaller fragment above this. (Fig. 5.)

Operation: The usual debridement of the superficial tissues was carried out while the depressed bone area was encircled with forceps and removed. There was a large amount of damaged cortical tissue as well as blood clot extruding from a long rent in the dura. The main trunk of the middle meningeal artery had been torn across. This was ligated by a silver clip. The blood clots and pulped brain were removed by suction and the wound closed without drainage.

After operation the patient had a slight



Fig. 20. Case xiii. Anteroposterior *x*-ray after second operation to illustrate replacement of large bone fragments.

temporary weakness of the left arm and leg which had cleared up at the time of her discharge. There remained, however, a definite astereognosis in the left hand. The postoperative scar is shown in Figure 6.

Report: May 9, 1927 (five months after injury). Seemed in perfect health. Has had two convulsions. No astereognosis of left hand, but slight hypesthesia. Normal mentally.

The case just cited illustrates, as did so many cases during the late war, that serious, penetrating wounds of the brain may be cleaned out thoroughly and sutured without drainage provided they are capable of being operated upon within ten to twelve hours after injury. The bone fragments would have been replaced ordinarily, but it was not deemed wise to take this hazard in a case in which brain damage had been considerable and since such a

large dural tear was present. The practically complete clearing up of neurological evidences of brain injury is interesting.



Fig. 21. Case xiii. Lateral x-ray after resorption of aerocele for comparison with Figure 19.

The following is an example of an extremely extensive area of depression with unusual operative difficulties:

Case vi. I. R. D., a girl of eleven, surgical no. 38205, was referred on February 11, 1931, by Dr. J. M. Fallon of Worcester, Mass

She had been in a motor accident on January 5, 1931, which was about a month before admission. She had struck the left side of her head and had bleeding from the nose and from both ears. She was unconscious for three days, being treated at a local hospital during that time. Following her injury there had been slight speech difficulty and certain indefinite mental changes including some loss of memory.

X-rays of the skull showed an immense comminuted and depressed fracture of the posterior, inferior portion of the left temporo-occipital region. (Fig. 7.)

Operation: February 13, 1931. After a skin flap was turned back the upper two-thirds of the depressed area were encircled by bone forceps and removed. When one of these pieces was lifted out a long spicule was seen projecting into the brain. As this was withdrawn, the greatest care being used, some adherent cortical vessels were torn which caused a serious hemorrhage before they could be controlled. This necessitated a small transfusion. The lower portion of the depressed



Fig. 22. Case xiii. Appearance of patient and operative site at time of discharge.

area was elevated without removal. As a final step some of the bone fragments removed from the upper portion of the depressed area were replaced. (Fig. 8.)

The child made an uncomplicated recovery and was discharged March 2, 1931. At this time she seemed perfectly normal in every way. She read, wrote and spoke without noticeable defect.

End-result (one year): April 6, 1932. A letter from the child's father stated that she was in excellent health and attending school regularly.

An example of complicating features necessitating a delayed operation is illustrated in the next case:

Case vii. C. D., a child of eight, surgical no. 34741, was referred by Dr. W. A. Bartlett of Manchester, N. H., on August 31, 1929.

This child had sustained a compound fracture of the right parietal region in a motor accident on February 20, 1929. The scalp wound had been sutured at a local hospital,

but broke down and a fungus cerebri developed with a cerebrospinal fluid leak. It was in this stage that I first saw the child in consultation. With the wound in this condition it was obviously inadvisable to attempt an operation upon the fragmented bone. Careful dressings, covering the fungus with gutta percha tissue, were advised in the hope that the fluid leak would stop and the fungus recede. At the end of six to eight weeks this was accomplished, but operation was postponed until some six months after the date of her accident.

X-ray at the time of her admission showed the old depressed fracture in the right parietal region with two loose fragments and a small bone defect. (Fig. 9.)

Operation: September 6, 1929. The old scar was excised and the loose fragments of bone removed, leaving an area of defect approximately 6 × 6 cm. This area was covered partially by replacement of the fragments.

End-result (two years): She made an uneventful convalescence, and in October 1931 her doctor reported that she was perfectly well although there was still a small area of skull defect.

In the next case there is illustrated what is perhaps the simplest method of dealing with a depressed fracture. This maneuver is not always applicable, and of course should not be attempted when fragments of inner table have penetrated through the dura.

Case viii. S. S., a girl seven years of age, surgical no. 27650, was referred on November 18, 1926, by Dr. C. H. Rapport of Chelsea, Mass.

She had fallen off a piazza three weeks before admission, landing on the left side of her head. She was unconscious for three hours, but seemed perfectly well after that. *X*-rays of the skull showed a linear and depressed fracture of the left frontoparietal region, the linear portion extending into the left orbit. (Fig. 10.)

Operation: November 26, 1926. The posterior depressed portion of the fracture was exposed by the usual skin flap, disclosing an oval area of depression 4 × 6 cm. in extent. There was very marked depression at the posterior end of this area. Consequently, a single burr opening was made above this region and a gutter extended by bone cutting forceps around the posterior end. It was then possible to get a blunt instrument under the depression, and when it was found that there were no spicules on the inner surface it was elevated back into its normal position. A single very slight nick in the dura was evident, but no damage to cortex.

End-result (two years): This child was discharged perfectly well on December 3, 1926. (Fig. 11.) Her mother reported on January 26, 1929, that she was very well in general but that she had some difficulty in concentrating on her studies.

Class vi. *Gunshot Wounds:* Patients with this type of head injury are seldom seen in civil life, but much was learned and written about them in the late war. For those interested in this subject the following articles by Gray,[3] Cushing,[4] de Martel[5] and Horrax[6] may be consulted. Any degree of damage may be encountered, from a simple grazing wound which barely furrows the skull to the severe penetrating wounds with the foreign body retained, or still worse, those which traverse the skull and its contents entirely. The essential features of treatment are to clean carefully the wound of entrance and that of exit if one be present by cutting out the soft tissues, after which the hole in the skull should be encircled with cutting forceps and removed. Bone fragments will be found to have been showered into the brain along the track of the missile, and these should be removed with the greatest gentleness and care by delicate forceps. Clots and pulped brain within the track may be sucked out with a syringe attached to a catheter, or they may be extruded by the patient's straining or coughing slightly. If the bullet is easily accessible it should likewise be removed, but more harm than good is done as a rule by attempting to get the ones which lie deeply in the brain. If left alone they will ordinarily cause no future difficulties.

The following case is the only one of this kind in our series during the past five years:

Case ix. R. P. Z., a boy of twelve, surgical No. 36885, was referred from the accident

room on July 13, 1930. Three hours before admission he had received a bullet wound (.22 cal.) in the right frontal region while playing around a rifle range. He was not rendered unconscious, and was perfectly rational when admitted, although highly nervous and excited. *X*-ray of the skull showed the wound of entrance in the right forehead, together with many retained fragments both of bone and of metal. (Fig. 12.)

Operation: July 13, 1930. The usual debridement of soft tissues and bone was accomplished without difficulty. Most of the indriven bone fragments along the track in the brain were also secured, but a severe hemorrhage from deep within the cortex prevented the finding of one or two of the bone pieces. This hemorrhage was controlled by a small piece of muscle which was placed within the track. No attempt was made to secure the metal fragments as they lay deep within the frontal lobe. The wound was left partly open directly over the hole in the bone.

The boy made an uneventful convalescence. One or two small fragments of bone were extruded through the wound, and the latter closed over completely.

End-result (one year): June, 1931. He has been perfectly well in every way since the accident and doing well in his regular class in school.

Complications

It is not within the scope of this article to deal with all the conditions which may be subsequent to slight or severe head injuries. Suffice it to say that I have picked out from among the cases which have formed the basis of the present study four of the more serious types of sequelae, allowing the brief protocol of each patient to tell its own story as to diagnosis and treatment.

Meningitis is no doubt the most feared as it is ordinarily the most fatal of all the complications of skull fractures. As a rule it develops fairly early, most frequently by way of a perforated drum membrane or fragmented accessory nasal sinus. In the case now to be cited, however, it came late in the course of a severe compound, comminuted fracture in the temporal region.

Case x. T. F. L., a woman twenty-one years of age, surgical No. 21233, was referred April 25, 1924, by Dr. C. K. Benson of Dedham, Mass.

She had been in a motor accident two days previously, at which time she was thrown forward hitting the right side of her head upon the screw which adjusts the windshield. This screw was driven through her skull, and brain tissue was found on the screw. She was taken at first to a local hospital where dressings were applied to her head and after being there two days was transferred to the Brigham Hospital in a semi-stuporous and quite irrational state.

Examination showed a lacerated wound of the right temporal region through which brain tissue the size of a hen's egg was extruding. Dried blood and hair were matted into the wound. There was an evident left hemiparesis and hemihypesthesia with loss of the superficial reflexes on the left side and an increase in the deep reflexes. There was also a right oculomotor palsy. *X*-rays showed a badly comminuted fracture of the right temporal bone with several loose fragments projecting into the intracranial cavity.

Operation was performed the day of admission. Radiating incisions were made from the margin of the wound and the dirty portions of scalp cut away. The protruding fungus was sucked off. All bone fragments were removed (Fig. 13) and the bone edge around the fracture excised with rongeurs for a distance of 1–2 cm. until fresh, unharmed dura was apparent. The whole wound was washed thoroughly with saline, while all necrotic, soft brain tissue was sucked away. The area was then covered with thin gutta percha tissue and left widely open.

A fungus of course developed but although the wound tended to clean up well the lateral ventricle was finally drawn out into the fungus and ruptured at the end of two weeks. A rubber tissue drain was inserted into the ventricle, the wound being dressed daily and always kept covered with smooth gutta percha over which a "doughnut" of cotton was placed to keep pressure off the fungus. The fungus granulated rapidly but a constant leakage of cerebrospinal fluid was present,

and on May 18 the patient's temperature rose to 104.3°F. and three days later she became dull again. It was thought that this was the end, but she rallied and her fever subsided. On June 9 she was very much better and the whole wound had granulated. Pinpoint grafts were applied to the area. (Fig. 14.) On June 28 she was up in a chair, and was discharged on July 11 at which time she was up and about the ward.

End-result (seven years): January 3, 1931. She had been having convulsions at intervals, but her general condition was quite good.

Brain Abscess: This is likely to be late in its development, and manifests itself as a rule first by symptoms of increased intracranial pressure or by evidences of localized brain involvement such as Jacksonian convulsions, progressive weakness of one side, mental disturbances, aphasia, etc. The treatment of a brain abscess varies greatly with many factors, particularly its situation and its depth from the surface. The articles of Eagleton,[7] Coleman,[8] Elsberg,[9] McKenzie,[10] and others may be consulted for details.

The following case illustrates some of the vicissitudes in the treatment of a post-traumatic brain abscess:

Case xi. D. DiM., a boy of seven, surgical No. 26177, was referred on April 17, 1926, by Dr. C. L. Craigin of Portland, Maine.

The child had been in a motor accident on February 7 of the same year, having had an operation upon his skull at a local hospital. Some pieces of bone were said to have been removed from the skull. Since this operation the wound had continued to drain pus; he had recently developed headaches and an increasing weakness of his left arm, together with focal seizures involving the left arm and leg.

Examination showed a left hemiparesis, the left pupil larger than the right, bilateral choked disks, and a fungus cerebri through the old operative scar over the right parietal region. (Fig. 15.) *X*-rays showed an operative bone defect under the area of fungus.

A needle was introduced through the fungus and 40 c.c. of pus evacuated from the underlying abscess. Four days later this process was repeated, and after a third evacuation it was possible to insert and leave in the cavity a small rubber catheter. The fungus increased in size for a time and a cerebrospinal fluid leak developed with the patient running a temperature up to 103°F. Finally the fungus granulated and subsided completely. His hemiparesis cleared up partially and he was discharged home July 26, 1926. (Fig. 16.)

The boy returned to the hospital on January 19, 1927, with a recurrence of headache and some protrusion of the former operative site. His hemiparesis was about the same as at the time of his discharge five months previous, but he had again developed choked disks. Three days after his admission his condition changed suddenly. He complained of intense pain in the back of his neck, became pale with rapid breathing, the protrusion on his head became tense, and within a few minutes he was semiconscious. A needle was introduced immediately through the bulging area and 60 c.c. of pus evacuated from the recurrent abscess. This improved his condition greatly and the process was repeated at two-day intervals for six days, the cavity being irrigated with Dakin's solution, and finally a rubber tissue drain was left in the cavity, and the wound dressed every two or three days. This method of treatment, however, was not found to be sufficient because of inadequate drainage.

Operation: March 31, 1927. The upper pole of the abscess was exposed over an area 2 by 3 cm., its contents evacuated, and the exposed area uncapped. The edges of the wall were then sutured to the subcutaneous tissues with several silk sutures, completely marsupializing the cavity. It was held wide open by gauze packed over rubber tissue.

Convalescence was uneventful after this final procedure, the wound granulating and healing completely so that he was discharged on June 4 walking about but with weakness of the left side most marked in the arm.

End-result (two years): April 8, 1929. A letter from the father said that the boy was in excellent general health and attending school regularly. Weakness of the left arm remained as upon discharge.

Subdural Hematoma: An uncommon late complication of any head injury, perhaps more frequent after the less severe types of trauma, is the slowly forming, encapsulated clot which lies between dura and

brain surface. In the literature this condition has been referred to frequently as "pachymeningitis hemorrhagica interna," a term which seems unfortunate not only because of its length but because it implies an inflammatory process when none is present. Putnam[11] reviewed the subject thoroughly in 1925.

The diagnosis of this condition may be made at times, or at least strongly suggested, by a history of skull trauma followed by an interval of several weeks of freedom from symptoms, and this free interval in turn followed by headaches which become extremely severe. When a subdural clot is either mildly or strongly suspected, it is our custom to make two burr openings in the skull, one on either side of the midline well back in the parieto-occipital region. The presence or absence of such a lesion may be demonstrated by a small incision through the dura. An osteoplastic flap is then turned down over the involved hemisphere and the hematoma together with its surrounding membrane removed.

The following is a typical example in practically all its aspects:

Case xii. M. C. S., a man of twenty-seven, surgical no. 23473, was referred on March 21, 1925, by Dr. J. J. Skirball of Boston.

Five weeks before admission he was struck in the left temple by a wrench. He was not rendered unconscious and continued work the rest of the day. One week after the injury he started to have headaches. These increased in severity until five days before admission when they became violent and were associated with vomiting and the development of diplopia. Examination showed anosmia, nystagmus to right and left, and optic disks with hazy borders. *X*-rays of the skull were negative.

Operations: March 28 and 30, 1925. On the former date bilateral taps were made on either side of the median line of the skull, disclosing a subdural hematoma on the left. On the second date a bone-flap was turned down over the left hemisphere, the dura reflected and the clot with its encircling membrane removed. A small rubber tissue drain was left.

The patient made an uneventful convalescence and was discharged April 18, 1925. (Fig. 17.)

End-result (six years): He has remained perfectly well, returning to work six weeks after his operation, and has worked regularly ever since. He has been seen from time to time up to the present date (November 15, 1931) and has continued perfectly well.

Pneumocephalus: The fourth and last of the complications of skull injuries which I have chosen to discuss are the intracranial pneumatoceles, namely collections of air within the cranial cavity resulting from a fracture. These collections may be extradural in some instances, but the more usual and certainly the more serious are those which lie within the brain itself. During the past year Rand[12] has reviewed this subject fully while reporting several of his personal cases. The commonest injury leading to this complication is a fracture through the frontal sinus with laceration of the dura at the tip of one or both frontal lobes. Following such an injury in which an intracranial pneumatocele has developed one can almost always obtain a history of the patient having coughed, sneezed or blown the nose rather violently. Sometimes during one of these incidents the patient may be aware of a sudden sensation inside the head. After this headaches are likely to develop, and if the air cavity increases to such size as to rupture into one of the ventricles a cerebrospinal rhinorrhea will ensue. This, of course, is a most serious happening, and unless the dural rent is promptly repaired meningitis is almost inevitable. In addition to the features just outlined, the diagnosis of pneumocephalus is made certain by stereoscopic *x*-rays of the skull which show distinctly the location, size and extent of the air cavity, together with its point of origin.

Although conservative treatment, by which is meant rest in bed under careful observation, may in some instances be sufficient to allow the air to absorb, operative closure of the dural laceration seems

without doubt to offer the best chance of forestalling infection. If the torn dura is easily accessible it may be sutured with fine silk, but when the tear lies well down over the tip of the frontal lobe it must be covered by a muscle graft. The latter is simply placed over the laceration and held down firmly for a minute or two until it adheres.

An excellent example of a large intracerebral pneumatocele which was treated by the above method is the following:

Case xiii. D. S., a young man twenty-one years of age, Surgical No. 29013, was brought in from the accident room at 6.30 A.M. on June 13, 1931. He had been thrown out of a motor car against a tree about an hour before admission. He was rendered unconscious immediately and was still in profound coma upon entrance. There were extensive lacerations and contusions over the right side of his face and head, both eyes being closed with ecchymotic edema. His cold, clammy skin, together with a rectal temperature of 96.2°F. were further evidences of severe shock. A large area of depressed skull fracture was palpable over the right forehead and *x*-rays showed some of the fracture lines entering the frontal sinus while others crossed the floor of the anterior fossa through the cribriform plate involving the sphenoid ridges and the roofs of both orbits. (Fig. 18.)

He was given the usual treatment for shock for three to four hours at the end of which time his condition was improved sufficiently for him to be operated upon.

Operation 1: June 13, 1931, 10 A.M. The fractured area was exposed by turning down a skin-flap over the right forehead. Two of the large bony fragments were temporarily removed and preserved in sterile gauze while some of the smaller fragments were permanently removed. The dura was widely lacerated in several places and masses of soft, contused brain tissue together with blood clots were extruding through the tears. These were washed away or sucked out. There was likewise great comminution of the frontal sinus with tearing of its mucous membrane. After a thorough cleaning up, the two largest bone fragments were replaced and the skin flap sutured back without drainage.

After this procedure he regained consciousness and rationality rather promptly and his convalescence was uneventful until July 2d, *on which day he sneezed and blew his nose rather hard.* He said later that at this time he "felt air bubble into my head." *X*-rays were taken the following day and showed a large intracerebral pneumatocele. (Fig. 19.) Because of its size it is quite probable that it may have been present longer than twenty-four hours and that the sneezing of the day before had merely increased its extent.

Operation 11: July 3, 1931. The old skin flap was re-elevated and the largest of the loose bone fragments again removed and placed in sterile gauze until the end of the operation. The tears in the dura in the upper portion of the field were healed firmly and new tissue had covered over the widely lacerated frontal sinus. Exploration was therefore carried down over the tip of the right frontal lobe and at its lowest portion, just over the cribriform plate, there was disclosed a round hole in the dura communicating with the pneumatocele. A muscle graft was taken from the patient's temporal muscle and held over the hole until firm adherence was established. No attempt was made to empty the air from the cavity since it was desired to have the frontal lobe and its covering dura come back into its place of close approximation against the skull, thus holding the muscle graft firmly over the hole. The large bone fragment was restored to its original position once more and the skin flap sutured without drainage (Fig. 20.)

The patient again made an uneventful recovery and *x*-rays taken at intervals showed that the air had been entirely absorbed by August 13, 1931. (Fig. 21.)

Report: March 15, 1932. He had up to this date remained perfectly well and had begun to resume his usual activities. (Fig. 22.)

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CERTAIN FACTORS IN THE OPERATIVE MORTALITY OF ACUTE APPENDICITIS*

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THE death rate for appendicitis in the United States in 1930 was 17 plus per 100,000 or a total of 17,687. In 1923 it was 22.3 per 100,000; in 1924, 17.3 per 100,000. The number of deaths attributed to appendicitis in the State of Illinois, in 1929, was 1298 or a death rate of 17.4 per 100,000 population.[1] The mortality from this cause in European countries is apparently much lower. In Paris, France, the death rate was 6.8 per 100,000 in 1929. The mortality in England and Wales for the same period was 7.1 per 100,000 population.[2] During the year 1926 seven European countries showed a death rate for appendicitis varying from 2 to 11 per 100,000 (Table 1).[3] Not only do our statistics on this condition compare unfavorably with those abroad but, according to McClure,[4] the death rate due to appendicitis in this country is actually increasing.

TABLE 1

APPENDICITIS DEATH RATE PER 100,000, INTERNATIONAL STATISTICS

Country	Year	Rate
United States	1927	15
Switzerland	1926	11
Scotland	1926	10
England and Wales	1926	7
England and Wales	1929	7 1
Germany	1926	7
Netherlands	1926	4
France	1925	3
Italy	1925	3
Spain	1926	3
Greece	1926	2

As shown in Table II the average operative mortality in acute appendicitis is about 5 per cent.

In an effort to throw some light on the factors influencing operative mortality the records of 2500 cases in which an appendectomy was performed at the Evanston Hospital in the last ten years were examined. For this study only those cases, 1138 in number, in which the diagnosis of acute appendicitis was confirmed by the pathologist were used.

TABLE II

MORTALITY IN ACUTE APPENDICITIS IN VARIOUS CLINICS

Author	Clinic	Cases	Deaths	Mortality (Per Cent)
Hunter, John B.[5]	London Hosp.	602	21	3 5
Christopher, F., and Jennings, W. K.	Coll. from Evanston Hosp.	1138	47	4 13
Wilensky, A. O.[6]	Collected	1765	86	4 9
Colp, R.[7]		2,841	148	5.2
Eliason, E. L., and Ferguson, L. K.[8]		675	37	5.4
Flint, E. R.[9] .	Leeds Gen. Inf.	1080	62	5 7
Bower, J. O.[10]	* Coll. from 27 Philadelphia Hosp.	5121	306	5 97
McClure, R. D.[4]	Ford Hosp.	940	61	6.5

* Mortality in these different hospitals varied from 1.64 to 11.29 per cent.

As indicated in Table III the gross mortality in 955 cases of acute, non-perforative appendicitis was 1.78 per cent. In the 269 of these cases in which drainage was employed the mortality (4.8 per cent) was over eight times that of the 686 cases in which drainage was not employed. The question suggests itself as to whether the drained cases offered a poorer prognosis at the time of operation. However, the diagnosis of acute, non-perforated appendicitis in all of these cases was established by the pathologist. While, no doubt, drainage was instituted in many cases because of the gangrenous appearance of the ap-

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pendix at operation, we know of many other instances, in this same series, where the appendix was under great tension, obviously gangrenous and associated with free fluid in the peritoneal cavity and in which no drains were used.

Table III

Acute appendicitis (non-perforative), Evanston Hospital, 1920–1931

A. Cases not drained	686	
Deaths	4	
Mortality	0 58	per cent
B. Cases drained	269	
Deaths	13	
Mortality	4 8	per cent
C. Total non-perforated cases	955	
Deaths	17	
Mortality	1 78	per cent

The gross mortality of the 183 cases in which perforation had occurred was 16.39 per cent. All of these cases except three were drained,* and in these three there was no mortality. The absence of mortality in perforated cases in which drainage was not used is suggestive, but the number of cases is too small to draw a definite conclusion.

Table IV

Acute appendicitis (perforated), Evanston Hospital, 1920–1931

A. Cases not drained	3	
Deaths	0	
Mortality	0 0	per cent
B. Cases drained	180	
Deaths	30	
Mortality	16 66	per cent
C. Total perforated cases	183	
Deaths	30	
Mortality	16 39	per cent

Of the 180 drained perforated cases the appendix was removed in 151 cases with a mortality of 17.88 per cent and not removed in 29 cases with a mortality of 10.34 per cent (Table v).

Where removal of the appendix involves handling of the intestine with the probability of dissemination of infection it may be better to omit removal and to be content with drainage.

Table V

Acute appendicitis (drained, perforated cases), Evanston Hospital, 1920–1931

A. Cases in which appendix was not removed	29	
Deaths	3	
Mortality	10 34	per cent
B. Cases in which appendix was removed	151	
Deaths	27	
Mortality	17.88	per cent
C. Total	180	
Deaths	30	
Mortality	16 66	per cent

A table was compiled which showed the results of operations for acute appendicitis for each surgeon at the Evanston Hospital who performed five or more operations during the previous ten years. The mortality and operating time were noted.

Table VI

Acute appendicitis, all classes, Evanston Hospital, 1920–1931

Surgeon	Total Cases	Average Op Time	Total Deaths	Av. Op Time Fatal Cases	Gross Mortality (Per cent)
A	234	34	14	36	5.98
B	207	39	9	34	4 34
C	115	26½	5	24	4 34
D	57	53	2	37	3 50
E	56	51	0	0	0 00
F	53	26	1		1 89
G	50	43	1		2 00
Total 7 operators	772		32		4 14
61 other operators	366		15		4 09
Grand total	1138		47		4 13

The two surgeons with the shortest average operating time, viz. twenty-six and twenty-six and one-half minutes, had a mortality of 1.89 per cent and 4.34 per cent respectively, while the two requiring the longest time, fifty-three and fifty-one minutes, had a death rate of 3.50 per cent and 0.00 per cent respectively. It would seem that skill, delicacy in handling the tissues, and judgment cannot be supplanted by mere speed *per se*.

* "Drained cases" were defined as those in which drains were placed in the peritoneal cavity.

CONCLUSIONS

From a study of the operative mortality of 1138 cases of acute appendicitis at the Evanston Hospital over the last ten years the following inferences may be drawn:

1. In the non-perforated cases of acute appendicitis the operative mortality where drainage was employed was more than eight times that of the cases where drainage was not employed.

2. It is suggested that in perforated cases of acute appendicitis which require drainage, and in which removal of the appendix requires much manipulation of the intestines, the removal of the appendix increases the mortality.

3. The mere speed with which the operation is performed has little influence upon the prognosis.

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* Continued from page 15.

A NEW FRACTURE, X-RAY AND ORTHOPEDIC TABLE

THE AUTHOR'S ORIGINAL ORTHOPEDIC TABLE REDESIGNED*

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AT the meeting of the American Orthopedic Association in Washington, in 1913, I presented my original orthopedic table. Since that time the question has often been asked, "Why can't the construction of the table be changed so that the *x*-ray can be used for making radiographs in cases of fracture and for reducing fractures under the fluoroscope?"

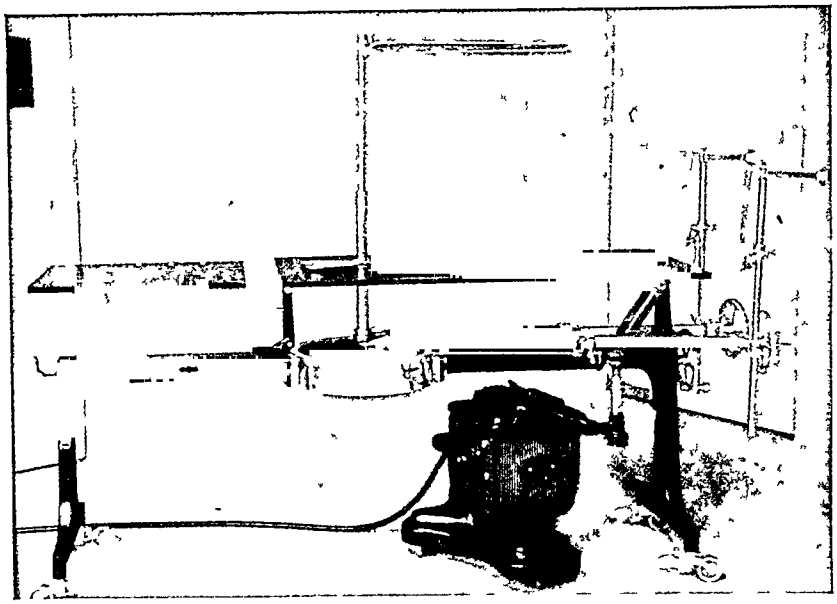


FIG 1. Author's table remodelled with floor shock-proof *x*-ray unit. All adjustments (gear for lowering top, traction control, and abduction lock) operate from foot of table and convenient to surgeon and assistants. No adjustment at center of table. *X*-ray unit here shown is flexible, handy and can be made to jack up for lateral exposures. This unit suitable for heavy duty. Can be operated continuously for one hour without overheating.

This did not seem practical until improvement had been made in *x*-ray apparatus. There was the double danger of exposure to the *x*-rays and contact with high tension wires. With the use of shock-proof apparatus there is no longer the danger of high voltage wires and refinements made in *x*-ray tubes make it possible to fluoroscope the bones of the extremities[1] with greater safety and effectiveness.

It is my belief that one of the coming developments in fracture treatment will be the use of the *x*-ray[2] in the reduction of fractures. Instead of the *x*-ray being used

[1] Recent trials have shown that one can see quite plainly the bones of the hip joint in an adult when the tube is placed at a distance of 15 to 20 inches. The tube in the new Model D unit has a small focal point suited for short distance exposures. It thus becomes possible to reduce fractures of the neck of the femur and dislocations of the hip, except in individuals with unusual thickness through the hips.

[2] The widespread use of the roentgen ray has been in the field of diagnosis Roentgen treatment is comparatively limited, limited to affections of the skin and neoplasms, chiefly. In fractures its use for the most part is for diagnosis of fracture and position of fragments. Its application in the treatment of fractures is still in its infancy.

* Submitted for publication August 18, 1932.

chiefly for radiographic diagnoses before and after reduction, it will be used to watch and guide reduction, and check

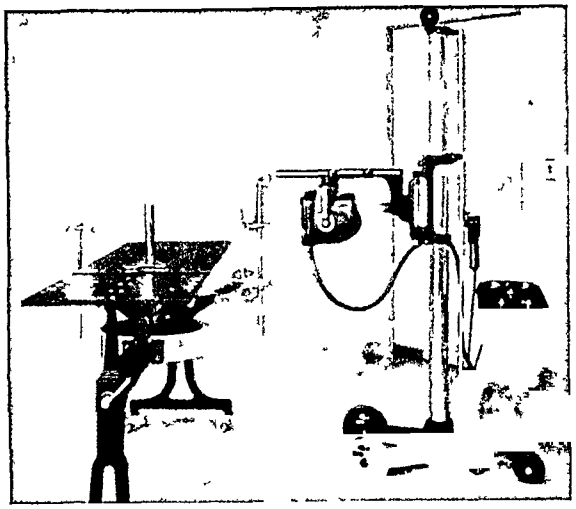


Fig. 2. Table with Model D x-ray unit having new small tube and small, compact tube head. This tube will do all that larger tube in Figure 1 can do. But duration of continuous exposure is short. This means interrupted exposures to permit cooling of tube between exposures. Interrupted fluoroscopy is more practical than continuous when limb is securely held in traction. It helps surgeon to regulate and measure exposure time, lessen chance of exceeding time allowance and prolong life of tube. This picture shows tube at side. Procedure has been to take one radiograph from above and one from side for diagnostic record before reduction. Tube is then placed below for purpose of fluoroscoping fracture during reduction. After reduction and immobilization radiographs are again taken in two planes. This tube has a very fine focal point making it suited to short distance exposure and fracture work.

the position of the bone ends during immobilization.[3]

To have to transfer a man with a major fracture to an *x*-ray table to obtain radiographs, then to a fracture table for reduction and immobilization, and then back again to the *x*-ray table is hard on the patient and all concerned. It would help if this could be done on one table and without so much effort. At the same time it would be an added advantage if the *x*-ray could be used to control and regulate manipulation and reduction.

[3] When plaster-of-Paris immobilization is used a radiograph can be taken at any stage without the patient being moved. Thick wet plaster makes fluoroscopy impractical.

To accomplish the above it is necessary to have a safe and efficient *x*-ray apparatus and an orthopedic table which does not



Fig. 3. Subject on table with one leg in traction and suspension and foot fastened to new foot piece. This consists of an adjustable sole piece for control of ankle flexion and a square horseshoe stirrup. Latter drops down when not in use, provides strong traction with little discomfort and regulates rotation of leg.

materially obstruct the passage of *x*-rays, which permits *x*-ray examinations from below, above and from the side, together with mechanical means for traction, manipulation and suspension of the leg and arm.

At the present time I feel we do not know very accurately the effect of traction, manipulation and posture in reducing fractures. These are carried out blindly. We see the results afterward in the radiographs. We do not see the bones during manipulation. We have not seen enough under the eye, studied visually which forces and motions are effectual and which ineffectual and why.

During the past year I have been at work designing a new table in such a way as to preserve the original features of the old table and incorporate improvements in traction, mechanical control of leg and arm, and make it possible and practical to *x*-ray a patient on the table.

This made it necessary to change the construction. As shown in Figure 1, one central horizontal beam with two end supports take the place of the old tubular framework. This provides a clear space below for the *x*-ray tube box. The leg

bars for traction and suspension are off-set to the inside 5 inches and centered on the perineal post.[4] In this way the leg bar is

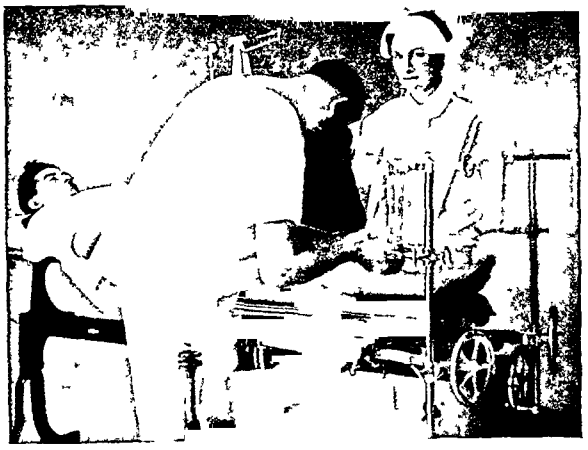


FIG. 4. Subject with leg in traction. Nurse holding box fluoroscope. Surgeon's hands free for manipulation to regulate traction and rotation. Or assistant can carry out manipulations under directions of surgeon. A longer telescoping cone from *x*-ray box to leg will still more confine rays. Lower section of top is split into two removable parts. One half is here shown removed when it is desired to remove all obstruction to rays.

not in the way when the *x*-ray tube is used from below. The abduction ring centered on the perineal post is large enough so that there is no obstruction when *x*-raying the bones of the hip.

The table top is new. It is composed of a wood fiber surfaced in a thin veneer of bakelite. This offers little obstruction to the *x*-ray and does not produce graining on radiographs. The bakelite veneer gives a clean smooth surface. The lower part of the table top is split into two removable sections (see Figs. 4 and 5).

An added feature is an armboard which makes it better for the handling of fractures of the humerus and of the bones of the forearm. There is better traction and support for reduction and immobilization under *x*-ray control.

[4] Dr. Albee makes a point on his table of having the leg bar pivot under the hip joint. That is a good idea, although it is not easy to determine accurately the joint center in different individuals. And it is not very important, because it is as easy to place the leg in abduction and then apply traction, as apply traction and then abduct. The former is more accurate and easier to do.

The new traction stirrup, as shown in Figure 6, is a distinct asset. I have tried 50 to 100 pounds' traction on my own ankle

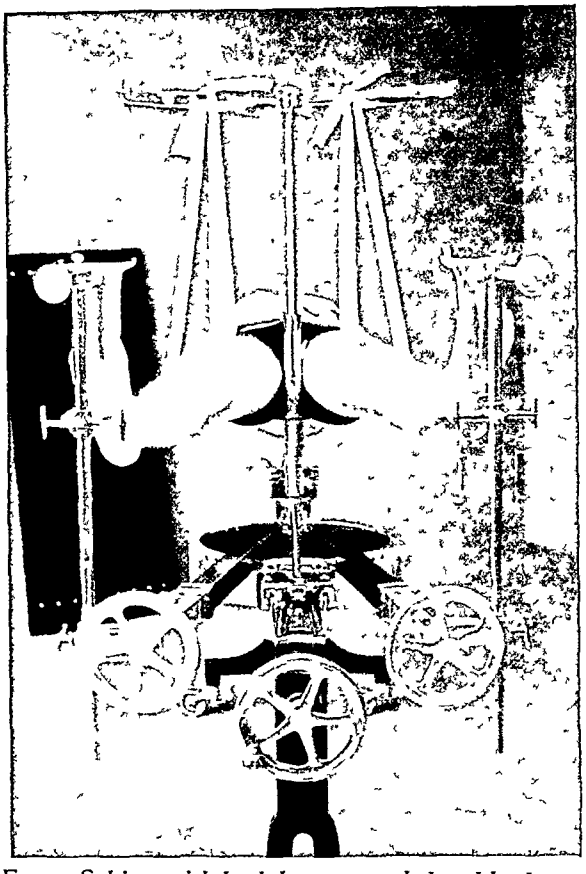


FIG. 5. Subject with both legs suspended and both sections of top removed. These are seen against wall. This photograph shows leg bars offset to inside so that bars do not obstruct *x*-rays when used from below. Left foot is in suspension without traction and traction stirrup is hanging down.

and it is remarkable what little discomfort it causes. Those who have seen and used it have been surprised how simple it is to apply and how effective the traction. Felt padding can be used under the traction band, but it is not usually necessary.

The leg bars are adjustable to a leg 22 inches from perineum to the sole of the foot up to one measuring 43 inches. This makes it suitable for a fairly young child as well as a tall man.

The perineal post is round. All in all, this has been found satisfactory. It is not easy to center the post in front of the

urethra and symphysis. A little more traction on one leg than the other forces the post to the opposite side. This is as it should be. The point of counter-pressure then comes on the ascending pubic ramus. It is natural to apply more traction to the injured leg, throwing the perineal post off center and against the pubic ramus instead of the symphysis.

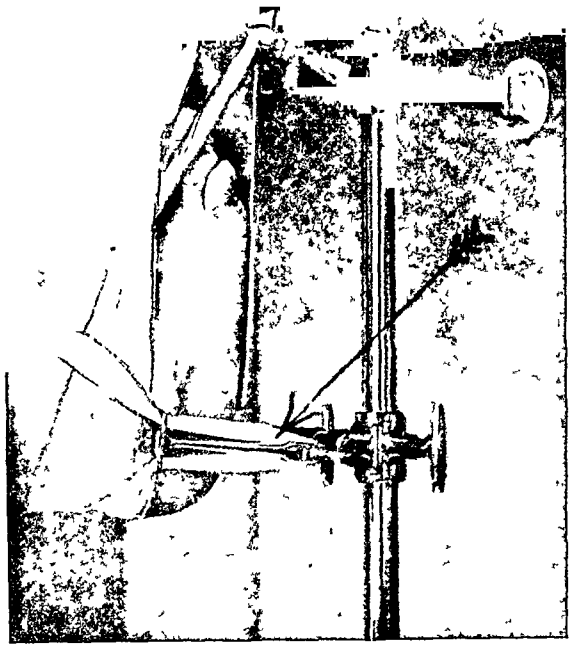


FIG. 6. Method of using traction stirrup. Object has been to find a method which was simple to apply, effective and easy to remove after foot is encased in plaster. Any traction device requires removal in order to be sure there is no undue pressure about ankle. To remove an anklet, Collins hitch or Delbet under wet plaster is not easy. In method shown above a gauze bandage is used. This is looped first over front of ankle, over bar below foot, then back of ankle. This is repeated until enough turns of bandage are made to give body and strength to loop. Turns do not cross anywhere so that when loop is cut at any point all tension is released, and bands can easily be removed. A half ring at upper end of stirrup takes place of a loop or a knot to release traction and remove stirrup after cast has been applied bands are cut as indicated by arrow or any place outside cast. Bands are withdrawn. Half rings are exposed. This requires some cutting of plaster. When freed each arm of stirrup can be hinged outward and then drops down out of way. Foot then slides off sole plate. A bandage loop under ankle as shown may or may not be used to hold foot suspended.

USE OF TABLE AND X-RAY IN THE TREATMENT OF FRACTURES

My experience in the reduction of fractures under the fluoroscope is limited. Generally speaking our knowledge is limited. There is much to be learned and time will demonstrate its possibilities and limitations.

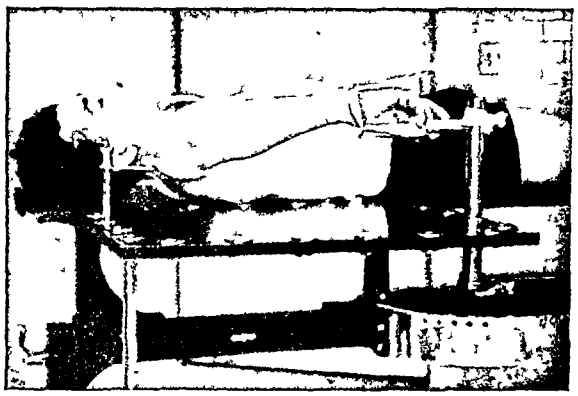


FIG. 7. Subject with armboard attached to side of table. Position for treatment of both bones of forearm, traction, reduction under x-ray and immobilization. A metal loop above elbow is used for counter traction with traction to a fixed post. Same form of traction stirrup is used for wrist as for ankle.

At present the plan of action in a fresh fracture has been somewhat as follows:

A man with a fracture of the tibia, for instance, is placed on the table. Traction is applied to the leg by means of the traction stirrup. Biplane radiographs are taken for permanent record and diagnosis. The fracture is reduced under the fluoroscope in day-light with the *x*-ray tube below. A nurse holds a box fluoroscope. The surgeon has his hands free and keeps his hands away from the fracture area. Reduction is made by regulating traction and by rotating and angulating fragments from the foot. This the surgeon does himself or in cooperation with an assistant.

Skeletal traction by pin, tongs or Kirschner wire can be substituted for the bandage-stirrup traction.

Reduction in difficult cases can be carried out by the subcutaneous introduc-

tion of a lever[5] to guide the fracture ends into position. This has been successfully carried out by Dr. C. M. Kerwin, of West Chester, Pa., especially in fractures of the forearm. He uses a dental elevator the handle of which gives strong leverage action. Recently I have been experimenting with a double handled lever.



Fig. 8. Traction and manipulation of fracture of both bones of forearm. Reduction under x-ray. Nurse holding fluoroscope and assistant regulating traction and rotation.



Fig. 9. Position for treatment of fracture of shaft of humerus. Assistant applying traction by grip on elbow or metal loop as in Figure 6 can be used. Counter traction by axillary post similar to perineal post (not visible in picture). Surgeon's hands free to cooperate with assistants in manipulations. This position suitable for immobilization in abduction splints or molded plaster. For immobilization in a plaster spica see Figures 9 and 10.

After reduction, temporary or permanent immobilization is carried out as the surgeon may choose.

Biplane radiographs are then taken to make the "before and after" record complete.

It will be possible, I believe, to introduce accurately a Smith-Peterson nail in fractures of the neck of the femur through a small incision made on the outer side of the greater trochanter. It is only necessary for some one to devise a guide so that the nail will penetrate directly into the head, not too far to the front or to the back. The depth of the head in relation to the trochanter will give the antero-posterior axis. The fluoroscope gives the other axis (the axis of neck in relation to shaft).

Early trials indicate that a small fluoroscopic screen is large enough because the fracture area is small. A small screen leaves the sides of the limb exposed.

It has been found that this fluoroscopic work can be done in daylight with a box fluoroscope. It is not necessary to wear a hood or accustom the eyes to the darkness

[5] This calls for as careful surgical technique as an open fracture operation.

except when fluoroscoping the region of the hip. Bone outlines show so sharply that the immediate accommodation of the eye is enough. Gross appearance is all that is necessary. The same fine bone and structural detail required in bone diagnosis is not necessary in reducing fractures.

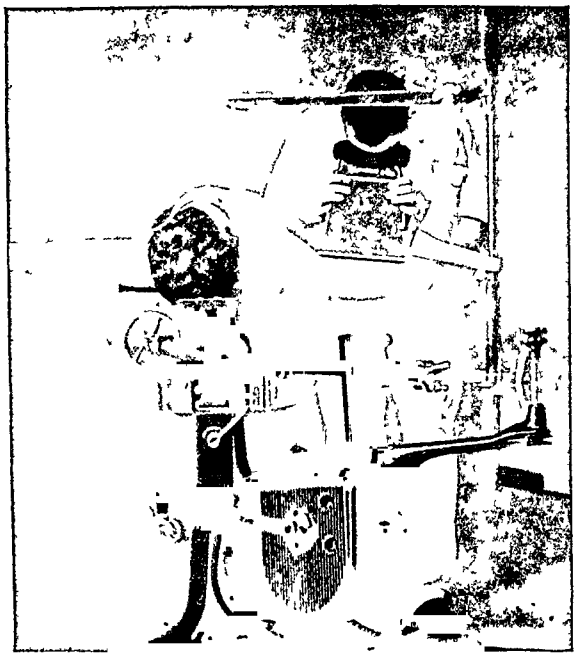


FIG. 10. Position for reduction of fracture of humerus under *x*-ray and application of plaster spica with arm in abduction. Nurse to hold fluoroscope omitted. Narrow board under spine. Counter traction on opposite arm by attendant or by bringing left leg bar into same position as right.

Biplane fluoroscopy for fracture reduction is not necessary or practical. The use of two screens and the need of the surgeon to view from above and then from the side is awkward and interferes with the reduction.

The biplane is necessary for radiographs, for still pictures, for views of fractures at rest. This is not so under the fluoroscope when the bone ends can be moved. When the bones are motionless it is difficult to tell in fractures with overriding which fragment is on top and which below. But when the bone ends are moved to one side or the other, it is not hard to make the distinction. In cases where a lever is introduced to facilitate reduction, or a long needle for local anesthesia, it is easy to see the position of the fragments.

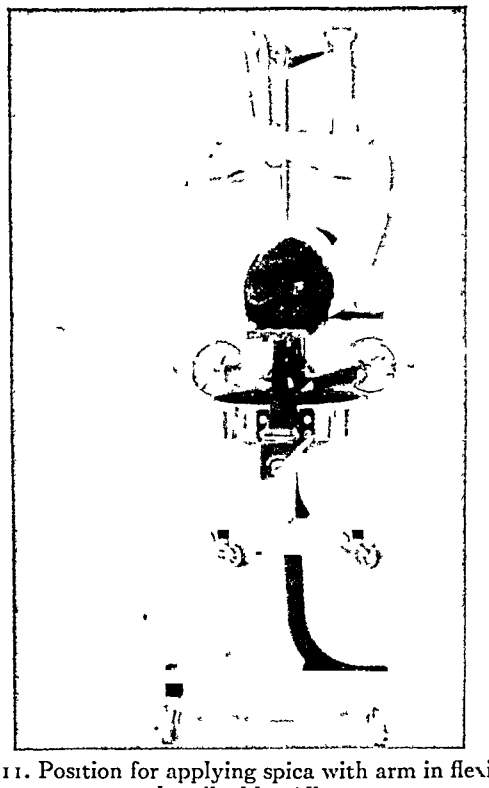


FIG. 11. Position for applying spica with arm in flexion, as described by Albee.

The ability to do everything necessary to reduce fractures in one plane makes it possible to direct the *x*-rays from below with the screen above. This, naturally, is the most convenient for the surgeon and attendants.

The biplane is necessary only for taking radiographic records.

What experience I have had in reducing fractures under the fluoroscope leads me to believe that with practice, the duration of *x*-ray exposure tends to become less. It is different from fluoroscopy in thoracic and abdominal diagnosis where the examiner can take his time, reasonably speaking. In the treatment of fractures the surgeon has a job to do and there is the natural desire to do it with little delay. With practice one can get accustomed to short, repeated illumination of the screen. It is

possible to install an automatic "on and off" switch in addition to the continuous foot switch. The operator then has his choice of either. To prevent over-exposure in each case, an alarm with cut-off can be controlled by clock mechanism.

With care and thought it is possible to make reduction of fracture under the *x*-ray reasonably safe because of:

The elimination of exposed high tension wires. The low milliamperage used. The use of a small diaphragm and small screen. The confining of the scattering rays by the use of an adjustable cone from tube box to the patient's leg. The exclusion of the surgeon's hands from the fracture area and from the direct rays. The use of mechanical and instrumental manipulation by the surgeon in place of the surgeon's hands to effect reduction.

I wish to thank Mr. S. Gwyn Scanlan of the Scanlan-Morris Company, and his chief engineer, Mr. W. W. Schaerff, for their interest and cooperation. To Mr. Schaerff belongs the credit for all the details of construction. His product is nothing less than a work of art. Also I extend my thanks to Mr. G. E. Wantz of the engineering department of the General Electric X-ray Corporation for his valuable and enthusiastic help.

To the Sisters of Charity of Glockner Hospital in Colorado Springs, and to Dr. W. P. McCrossin, attending surgeon, I am indebted for the opportunity to use this apparatus on actual cases.

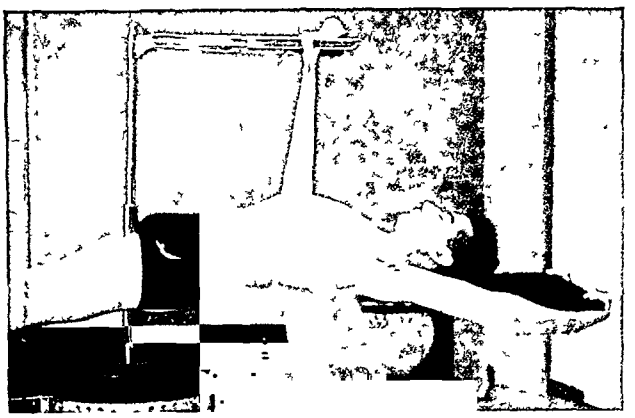


Fig. 12. Position for applying plaster jacket. The shoulder support adjusts to fit a small child or a grown adult.

PROBLEMS IN THE TREATMENT OF VARICOSE VEINS*

GEZA DE TAKATS, M.D., F.A.C.S.

CHICAGO

THE treatment of varicose veins has attracted such wide interest both among the practicing physicians and the public, that an unbiased discussion of its present status seems timely. It is characteristic of the history of any therapeutic procedure that, following a stage of initial enthusiasm, a second stage of critical reaction follows, during which the entire treatment may be discarded. Only if there is real value in the new methods, only if it means real progress, will they become part of our therapeutic measures.

The history of the treatment of varicose veins shows that vein ligations were already described and practiced by Celsus, that bandaging and vein-trusses were well known to the early pioneers of medicine, and that injections into varicose veins were introduced shortly after the invention of the hypodermic syringe as a natural sequence to the knowledge that an attack of thrombosis may cure varicose veins.

The last ten years, however, have brought a renewed interest in this subject and never before have so many patients been treated by so many physicians. It has become office practice in the hands of the general practitioner. The method of injections has been described, demonstrated at conventions, at county medical societies, discussed in the newspapers. Pharmaceutical houses have inundated physicians with more or less well-written pamphlets and recommendations to use this or that drug with this or that technique. A great deal of confusion arose. The American Medical Association, in order to create a clearing house and help disseminate simple and safe methods, authorized the formation of a Committee on Varicose Veins under my chairmanship, which for two years has organized educational exhibits at the A. M. A. conventions and issued pamphlets to the physicians.[1] The material of these exhibits is assembled at headquarters and may be loaned to state medical societies for exhibition purposes.

The contraindications to the injection treatment of varicose veins, the circulatory tests, the solutions used for injections, the technique, the prevention of tissue necrosis, the management of chronic leg ulcer which is so often on a varicose basis have become fairly well standardized and do not need elucidation. Instead, I have selected certain problems in the treatment of veins, which have received very little discussion in the literature in spite of their importance.

RESTING INFECTION IN VARICOSE VEINS[2]

That varicose veins are frequently infected is generally known. In a group of 31 cases of acute phlebitis in varicose veins, 6 could be traced in all probability to teeth and tonsils, 5 followed acute respiratory infections, 3 were precipitated following operations with prolonged immobilization and in 17 cases no definite source of infection could be found, but they were obviously blood-borne as no local infection was present. The importance of infection in varicose veins is obvious. It constitutes the most important contraindication to the injection treatment. No one would think of treating acutely infected veins with injections. Of far greater significance, however, is the type of vein which harbors bacteria but does not show any evidence of infection at a superficial examination. It has seemed to me, that of all the discomfort and untoward results that may follow

* Address given before the Rochester Academy of Medicine, Rochester, N. Y., December 3, 1931. From the Department of Surgery, Northwestern University Medical School.

injection treatment, the activation of slumbering infection is the most important factor and the least emphasized. The analysis of embolic deaths following injection treatment show that invariably two factors are present: an induced phlebitis, and a prolonged immobilization in bed. If one can avoid the former, the latter will not have to be resorted to.

In the course of ambulatory ligations, which we will discuss a little later, sections of the saphenous vein were placed in suitable media and studied by Dr. Arthur Kendall. Of fifty-eight cultures made, over one-half were positive and definitely not due to contaminations. It must be remembered that these veins were ligated in the absence of any clinical infection so that we were really dealing with supposedly non-infected veins. The bacteria grew very slowly and the cultures were frequently negative up to a week or ten days, indicating the sluggish growth of these hibernating organisms.

It seems certain then, that a large percentage of varicose veins harbor bacteria. In our 1500 patients treated in the Varicose Vein Clinic of Northwestern University and in my own private and consulting practice, 25 cases of acute phlebitis have been seen to follow injection treatment. These cases only include massive thrombophlebitides, extending from the site of infection up to the groin, with a marked periphlebitic edema. It is quite a characteristic syndrome. At the time of injection there is no gross evidence of inflammation. From two to seven days following the injection, the patient returns complaining of great pain and often, although not always, a rise in temperature is present. The injected vein and a large segment above it is thrombosed, surrounded by a periphlebitic exudate. The skin is red and hot. Frequently, if the valves are insufficient, the clot extends to the femoral junction.

Prolonged immobilization is not advisable. When the temperature drops to normal, the patients are allowed to get up. The legs are strapped to a point which extends above the upper limit of the clot. We have not lost one such case through embolism. As stated before, embolism following injection treatment is due to a soft, infected thrombus and the favoring influence of prolonged immobilization. If one could avoid the injection of veins that harbor resting bacteria, a most significant danger could be eliminated.

Recognition of Resting Infection. The history of an acute phlebitis should make one suspicious of a possible flare-up. Curiously enough, patients who have gone through an acute attack do not show marked reactions following vein ligation or injections and the same is true of chronic varicose ulcers—the acutely infected ones naturally being treated conservatively. The absence of an acute flare-up in these two groups strongly suggests the development of a local immunity of the veins to the preceding infection. On inspection, such veins do not show any obvious inflammatory reaction such as hyperemia or edema, but sometimes a residual pigmentation is present owing to an old hemorrhagic exudate. After emptying the veins by elevation, the walls are thickened, painful on pressure and small phleboliths are palpable. The most important finding is a rise in skin-temperature above such veins, which can be easily detected with the palmar surface of the hand and exactly determined by measurements with a skin-thermometer. A difference of 3° to 4° as compared with symmetrical regions on the other leg is not uncommon.

There are no systemic changes in these slumbering infections. Temperature and leucocyte count are normal. Much more fruitful seemed the application of certain provocative measures which served to produce a mild activation of the resting infection. I offer three harmless procedures, the first of which can be used without any special equipment.

A simple puncture of the vein is the mildest possible trauma and yet I have

seen it to be followed by a patchy thrombophlebitis in a suspicious vein. The administration of diathermy over the suspicious and another control area for five minutes, gives a marked difference in temperature when observed after twenty-four hours. The most exact data are obtained, however, by applying a ⅙ skin erythema dose of *x*-ray with deep filtration (90–100 R) to the questionable area and to a control area. The rise in temperature is best observed in four hours and is more pronounced over the area with resting infection.[2]

While a negative response to such provocative measures may not exclude "resting infection," a positive result is an important danger signal. It contraindicates injection treatment. The provocative venous punctures or the injection of a few drops of mildly irritating substances such as 50 per cent dextrose solution can be carried out without any special equipment.

Treatment of Resting Infection. When the temperature changes and provocative measures have made the diagnosis of resting infection, first a careful search for obvious foci must be made. The removal of such foci may temporarily aggravate the latent phlebitis. It is more difficult or quite impossible to eliminate pelvic infections, infected thrombi in pelvic veins, which follow childbirth or miscarriages and continuously reinfect the veins of the lower extremities. One such case has been observed for a long time. Every change of weather, every slight exertion, would light up small periphlebitic patches and, at the same time, the thrombosed veins in the broad ligament became painful to the touch.

Repeated doses of *x*-ray for resistant infections, with supportive Unna's casts and mild parenteral protein therapy offer possibilities. In 5 well-observed cases it was possible to clear up the residual infection, so that later injections could be given without any reaction.

The recognition of resting infection, then, is one of the most important safeguards in avoiding untoward reactions.

Patency of Deep Venous Circulation

A second problem which is more often discussed but very little understood, is the patency of deep venous circulation. Every paper, every instruction starts out with the warning not to inject varicose veins in the presence of deep venous obstruction. For years I have been testing for deep venous obstruction with the test of Perthes and, while it may point sometimes to an increased venous pressure in the deep veins, the answer is never convincingly definite.

Let us examine what happens after a deep venous thrombosis. The clot is usually in the external iliac vein, may of course be in the common iliac and hypogastric veins, and may extend down into the femoral. Experimentally, Zimmermann and I[3] have shown that the extent of edema or the possibility of collateral circulation will depend on the extent of the clot to the periphery. That is to say, that there may be "silent clots" in the pelvis without any edema of the lower extremities. That veins will dilate on the lower extremities without the true history of a "milk leg" is not infrequently observed. The venous obstruction in the pelvis calls for collateral circulation. But how long does the venous obstruction persist? The clot, unless it is very long, is reabsorbed and becomes canalized. The vein becomes patent again but there are two definite changes compared with the normal status. First, there is a new collateral network which is now superfluous and, second, there is a deep valvular insufficiency. Thus the important femoral valve which has been described and commented on by Turner Warwick[4] becomes incompetent. We are dealing now, not with a deep venous obstruction, but with a deep venous valvular insufficiency which resembles the congenital weakness or absence of the femoral valve on a hereditary basis. In such patients the hydrostatic

pressure and also sudden changes in abdominal pressure, that occur during straining, coughing, are all transmitted into the saphenous system and cause many communicating valves to become incompetent.

The injection treatment in such cases is not contraindicated, unless there is danger of activating the original phlebitis. There is no danger of shutting off too many venous channels. But the results of the treatment are not always permanent. The increased deep venous pressure may canalize the thrombi or the obliteration does not take place at all.

The recognition of this type of patient is based on (1) strong impulses at the site of saphenofemoral junction or at perforating branches, when the patient is asked to cough; (2) multiple valvular insufficiency of the perforating veins which is recognized by the fact that, in spite of a saphenous compression, the veins fill up suddenly when the patient lowers the leg from an elevated position. Just how we can improve the results of our treatment in this group will be discussed under the next heading.

RECURRENCES

The percentage of recurrences after injection treatment is the third important problem. Quint and I[5] published our findings in 1930 and reported a total of little over 10 per cent recurrences. Nelson Howard and his co-workers[6] at Stanford University have reported the astonishing percentage of 98 per cent recurrences following injection treatment. Outside of these two reports no careful follow-up records have become known, most authors writing enthusiastic articles on immediate results. Now it has been well known from older statistics on operative results, that recurrences increase with every year; that the end of the first year most recurrences are present, but that they increase up to the fifth year.

Vein ligations have been practiced for over a century with the distinct view of cutting off the column of blood from above and Trendelenburg advised saphenous vein ligation to protect the saphenous system from variations of intra-abdominal pressure. Yet a combination of vein ligations with injections on ambulatory patients has not been tried on a larger scale. Quillin and I[7] are reporting on 200 ambulatory vein ligations combined with injections. If the high ligation of the saphenous vein is done with proper indications and proper technique, a recurrence of 0.6 per cent was found. This one recurrence was due to an incompetent femoral valve, a condition that we have overlooked entirely up to the present time and that we believe to be almost uncontrollable.

On the basis of the 200 ambulatory vein ligations, I feel that this procedure has a very definite place in the treatment of varicose veins. The high saphenous ligation protects the injected veins from fluctuations of abdominal and hydrostatic pressure, thus preventing canalization of thrombi. It also reduces the necessary number of injections and places the occluding ligature close to the saphenofemoral junction. By insisting on a short proximal stump and an ambulatory management, we have no embolism to report in the entire series.

Our indications for this procedure are summed up as follows: (1) when there is valvular incompetence of the long saphenous vein above the lower third of the thigh; (2) when there are a few large incompetent communications between deep and superficial system, which can be ligated, and (3) in case of an ascending thrombophlebitis of the saphenous vein.

The operation is contraindicated (1) when the vein is not involved above the knee, and (2) when there is evidence of insufficient deep venous return owing to an old deep phlebitis or deep valvular insufficiency.

The most difficult type of case is the one which shows multiple incompetent communicating veins and an involvement of the long saphenous vein to the groin. Formerly we advocated a high ligation followed by a radical excision for this type,

under spinal anesthesia.[5] On the basis of more recent experience, it may be possible that these patients can be equally well treated by multiple vein ligations followed by injections. The ligatures must be placed on the perforating branches which have to be localized before the operation, and not on the saphenous vein itself, as otherwise an early recurrence will occur.

I have called attention to three problems that must present themselves to every physician who uses the injection treatment: the problems of activating infection, the problem of the deep venous return and the problem of recurrences. The injection treatment itself is fairly well standardized and does not have to be discussed here. But, unless one is on the lookout for complications or insufficient results, this otherwise excellent method will again sink into oblivion. It is with this object in mind, that I brought these problems before you.

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RELIABILITY OF CANCER STATISTICS*

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HARRISBURG, PA.

STATISTICAL evidence is exact when completely collected and correctly interpreted. Figures can be juggled to support any assertion, but that is not statistical evidence. The careless use or the incomplete collection of figures misleads to an extent as to seriously impair any investigation. Most persons who write or read statistical reports are acquainted with the errors of human judgment and the unavoidable errors in computations. Comparisons must be of large numbers to have value, and ratios that are nearly the same should be considered as equal. Minor differences are unimportant. Rates beyond the first decimal are probably of no value. A speaker who gives rates should use round numbers and omit the decimals so that the hearers will be more apt to remember the principal figures. Figures which have no practical value should be omitted. In many instances questions as to correctness of statistics would be avoided if the sources or the methods of obtaining the figures were given.

The figures in reports frequently give only half of the whole truth. They may mislead without intent. Often one reads the number of persons of a certain occupation who have cancer, but such an assertion, without giving rates, has no value. It merely indicates that some people in the neighborhood follow that occupation, and some have cancer. A survey accounts for certain conditions but never mentions the alternatives which are just as important. A spot map shows the location of a certain disease and is useful as far as it goes, but may be remiss in not explaining the conditions in the blank areas where circumstances may make it impossible to have the disease. Comparisons of things which are not comparable are attempted but the reader swallows the assertion and lets it go at that.

Mortality statistics, as usually used, are tabulated from the punch-card records made from death certificates. The punch-card has room for only certain information and obviously it is impossible for every disease or even every part of the body affected by cancer to be recorded separately on the punch-card. Groupings must be formed and the best effort has been made to have the groupings of diseases and of organs made in the most scientific and practical way. These groupings are useful for many purposes but not for every occasion. Numerous attempts are made to utilize them for purposes for which they were not intended, to prove what they will not prove.

Without quoting any authors for fear of being misjudged, a few illustrations will be offered to support these assertions. The figures were obtained by special tabulations from Pennsylvania death certificates for one year and from federal records for that state. The Pennsylvania figures are the tabulations of 9283 cases of malignancy found among 120,000 death certificates. The information secured is not indicated on the regular punch-cards.

Attempts are frequently made to report statistically the duration of the development of cancer and the postoperative length of life. When these figures are the result of careful clinical study they probably are as dependable as it is possible to determine. But when they are collected from death certificates, as has been done, they probably have little if any value. Unless it is obvious, it may be an advantage to state how the records of duration were compiled. The inaccuracies of these figures when obtained from death certifi-

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cates may be illustrated by our investigation. An examination of the death certificates of cancer cases showed that on 22 per cent of many hundred certificates the "duration" of the cause of death exactly corresponded to the two dates showing the length of treatment. To quote the duration of illness as given on death certificates, therefore, is very misleading. There was also a preponderating guess that the duration of the disease was one year, or similar designation.

The postoperative length of life is most reliably obtained by a follow-up of hospital cases rather than only by tabulations from death certificates. Postoperative mortality is not operative mortality, nor hospital mortality. The term operative mortality should be confined to deaths resulting as a complication of an operation. Death certificates do not always record whether or not an operation was done, and when; as an example, among 810 certificates for cancer of the breast only 210 gave a date of operation. What are reported as operative deaths should be restricted to those deaths directly attributable to the operation or to complications indirectly connected with the operation and inevitable deaths which occurred within a given time (as seven days). Examples of such cases would be postoperative pneumonia, cardiac collapse, shock and hemorrhage and intestinal obstruction (with intestinal cancer).

Women are said to have more cancers than men, a meaningless statement. Comparisons are also made of the death rates of the sexes as shown by governmental figures of malignant diseases of the stomach and liver, and of other groups of organs. More than fifty hospitals reported the sex of their patients and showed that 130 men and 68 women were treated for cancer of the stomach; 20 men and 34 women for primary cancer of the liver; 3 men and 11 women for cancer of the gall bladder; and 12 men and 5 women for cancer of the esophagus. Except possibly for cancer of the stomach, lip and skin, sex probably has nothing to do with cancer of organs possessed by both sexes, and a comparison of total cancers is odious.

The increase of cancer has been both asserted and denied. The only just investigation of this subject, I believe, can be made of the only statistical group which contains a single organ, the breast, and should include adjusted rates of only the oft-termed cancer age. The state and federal tabulated records of cancer of the breast can be used for such a study as they contain carcinoma and sarcoma of only the breast, and no other organs are involved.

The number of deaths from cancer of the breast is increasing twice as fast as the population of Pennsylvania. During 1927 the age groups 40 to 59 contained 47 per cent of the deaths from cancer of the breast, and 45 to 64 years contained 51 per cent. The federal statistics show the death rate from cancer of the breast increased in the 40–49 age group from 26.8 in 1910 to 32.7 in 1920, per 100,000 women of that age group; in the 50–55 year group the decennial increase was from 40.4 to 55.5; and in the 60–69 year group the death rate increase in ten years was from 61.0 to 65.0. Specific figures if obtained by similar methods would definitely indicate the actual trend in a death rate, but crude death rates, especially when applied to groups of diseases or of organs, indicate very little of value. We know definitely, therefore, that cancer of the breast shows an increasing death rate in Pennsylvania.

A similar study to determine an increase of deaths from cancer of the stomach or of the uterus or lungs would require (by a study of the original death certificates) a separation of the deaths from cancer of the particular organ from all the others in the group. There may be an increase in deaths from cancer of the female generative organs, but because that group contains many more organs or involved tissues than the uterus, and there are so many possible causative factors, any statement of an increase has no special

value. To determine the increase of gastric cancers it would be necessary to separate the primary cancers of the liver and gall bladder, and also all the cancers from the fauces to the lower end of the esophagus from the group. This can be done only from the original certificates.

The increase in the diagnosed cases of cancer can scarcely be credited to a betterment of facilities or technique for diagnosis. In the 1842 fatal cases of cancer of the stomach the means of diagnosis was recorded for 826; clinical diagnosis in 40 per cent of the 826 cases, laboratory analysis 4 per cent, *x*-ray diagnosis 22 per cent; the rest were diagnosed at operation or discovered by autopsy. In the hepatic cancers dependence was placed upon clinical suspicion in 60 per cent and an *x*-ray diagnosis was made in only 8 per cent.

While the number of deaths from malignancy of the stomach and liver has markedly increased, the deaths from undefined diseases of the stomach have decreased only slightly. To what extent the alleged diagnostic facilities are utilized may be inferred from the reports of 139 hospitals which are equipped with *x*-ray apparatus. At these hospitals, of 130 persons stated to have died of intestinal or peritoneal carcinoma, 70 were in the hospital four or more days, and *x*-ray diagnosis was attempted in only 4 of these patients, the remaining 66 being subjected to abdominal operation without every available diagnostic aid being utilized. All of these suggest, with other similar records, that, in the mass, increased diagnostic facilities are not the effective factor in the increase of recorded cases of cancer.

An increase in cancer deaths shows the need of earlier and better treatment; an increase in cancer shows the need of more knowledge of its causative influences.

Clinics, hospitals or surgeons reporting an increase of a particular disease among their patients make a statement of their experience only, and this has no bearing whatever on the condition of their city or state. Cases are not equally divided among hospitals. Three large hospitals in Philadelphia, as an example, had only 20 per cent of the total gastric cancers of 22 hospitals, but received 79 per cent of the cases of cancer of the esophagus during the year. One of the largest hospitals in the state during six years showed a decrease in lip and tongue cancers, mouth cases showed no change, and the larynx cases doubled; stomach cases quadrupled, rectal cancers increased one-half and cervical one-sixth. These simply show an experience and do not indicate any trend of cancer development.

The increase of deaths from all malignant disease of all organs is undoubtedly a fact, but, according to Ewing, we do not care to know the changes in the totals of all cancers but only those of particular organs. An increase in deaths from all violence means little, but to know there is an increase from one certain form of violence or accident has great significance. The total death rates from all malignant diseases from 1911 to 1930 in Pennsylvania gave the following five year averages: 71.6, 80.3, 89.5 and 98.4. This shows an undoubted increase, but the information is of little practical value. If the group of malignant disease of the stomach and liver is studied for the ten year age group showing the greatest number of deaths, 60 to 69 years, the resulting rates: 189.3 in 1910 and 225.8 in 1920, may create an interest, limited, however, because we do not know to what extent carcinoma of the stomach controlled these rates of a group of organs.

The deaths from cancer of the stomach as tabulated from state or federal death records are the group of deaths classed as from "malignant tumors of the stomach and liver." Many writers accept and quote these figures as representing cancer of the stomach. This group, however, includes deaths from carcinoma, from all the multiple forms of sarcoma and endothelioma. The organs included in the group comprise the fauces, throat, pharynx, esophagus,

stomach, including the pylorus, the liver, gall bladder, bile ducts and gall duct. The person who discusses or writes upon cancer of the stomach must consider these influencing variables if he utilizes the tabulated records of cities, states or governments. To what extent the extraneous cases included in this group will affect the general case, morbidity or death rate from carcinoma of the stomach cannot be herein determined. Suffice it to state, however, that the state records used as an example in this paper showed for the one year the following deaths which have not been heretofore published:

Carcinoma or cancer of the esophagus 132, stomach 1960, pylorus 144, liver 774 (including only 2 having the defining word "primary"), gall bladder 114; and sarcoma of the esophagus 1, stomach 10, liver 20. Not knowing how many of the liver cases were primary, we are not justified in drawing conclusions, although the crude death rate from this collective group is 33.0; from carcinoma or cancer of the stomach and pylorus 22.0; from the liver (many of which cases were doubtless metastatic), 8.1. The group rate, excluding the sarcomas, is 32.7. The group, therefore, does not represent gastric carcinoma. There were 45 deaths from cancer of the mouth and 5 designated as sarcoma. These were not included in the stomach group No. 44, but if half of them were faucial or pharyngeal (as seems likely) they should have been so described and then would have been grouped with the gastric deaths, thus altering the death rate only 0.3.

These examples show the many factors which should be considered when reporting on group rates. It is not likely that the same influence will produce cancer equally in all these organs. While cancer of the stomach and pylorus may increase, the esophageal carcinomas may decrease and adversely affect the combined death rates. Yet, reporters remark that gastric cancer is on the increase and also they make comparison of rates which differ in only minute fractions.

To what extent cancer of the liver influences the statistics of malignancy of the stomach and liver may be suggested by 96 hospitals which reported treating 681 cases of cancer of the stomach and 151 primary cases of cancer of the liver. Cancer of the liver thus comprised over 18 per cent of the group of cases, if these figures are accepted. The deaths of these cases within the hospitals represented 15 per cent of the stomach and 44 per cent of the hepatic cancer cases. Since gastric carcinoma is more amenable to operation or other treatment than is primary cancer of the liver, a very serious error is apparent when death rates of this group of cancers of the stomach and liver are quoted as representing cancer of the stomach.

Hospital records cannot indicate rates in relation to the general population. Only three-fourths of the cancer cases treated in 49 Philadelphia hospitals came from that city, and only 41 per cent of the deaths from cancer in Philadelphia that particular year occurred in the hospitals. These hospitals reported having 554 deaths from cancer, yet the death certificates from these same hospitals showed 682 deaths from cancer that year. Many death certificates of patients dying in hospitals do not indicate that the patients had been in any hospital. The inaccuracies on death certificates are notorious. The records kept by hospitals vary greatly, some are as complete and accurate as it is possible to make them, while others are too inaccurate or incomplete to have much value. Another factor to reckon with is the fact that the International Classification of the Causes of Death does not agree with the lethal causes as agreed upon by hospital authorities and records.

The questionnaire is frequently the only available method for collecting statistics, but because of the great numbers of questionnaires inflicted on busy people the system has become a nuisance. Many persons return wild guesses as their replies and these very seriously damage an important investigation. A brief ques-

tionnaire sent to the hospitals of Pennsylvania included a question of the number of deaths they had from cancer during the year. Their replies gave a total of just 26 per cent fewer deaths than the death certificates from these same hospitals showed for the same period. Thirty-five hospitals claimed fewer deaths and 41 admitted more deaths than could be checked on their death certificates. The hospitals reported receiving 681 cases of cancer of the stomach and 151 primary hepatic cancer cases. Many surgeons may doubt the prevalence of primary cancer of the liver as shown in those figures. Various other discrepancies were revealed by the questionnaire, although several large hospitals went to great trouble to furnish extensive reports. The questionnaire is not an infallible method of gathering statistics. It has limitations and before reporting its result the possible discrepancies should be sought and reported. The questionnaire should be brief, unmistakable in meaning, easy to answer; should omit all questions not absolutely necessary, and should not be employed if it can be avoided.

Let us then be more careful of our statistics; compare comparable factors; give definitions which define; report figures which correctly confirm, deny or explain conditions. Let us determine as far as possible the cause of the differences of figures or rates of similar conditions; be not too critical of another person's records which are correctly shown, but explain the conditions under which our records were obtained and what locality or condition they represent. We should publish statistics which others may understand and employ; use rates adjusted to correctly interpret a definite subject of a definitely involved group of persons; omit comparisons of groups which are irregularly influenced by various factors which cannot be eliminated and which are detrimental to the study in hand; and constantly endeavor to improve our records in completeness and accuracy.

THE WELL-LEG COUNTERTRACTION METHOD

DETAILS OF TECHNIQUE*

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FRACTURES of the lower extremity and the pelvis can now be treated by a new method of skeletal traction which, though simpler in application, obtains better results. The principle depends upon the utilization of the well leg alone for countertraction. As neither ropes nor weights have any part in the reduction process, the well-leg splint by means of its unique leverage attachment makes possible the combination of skeletal traction with well-leg countertraction (Fig. 2). The construction of the splint is so perfected that, once set in operation, disarrangement is impossible, even with wheel chair ambulation. This method reduces essentially the amount of after-care, and provides a safe mode of handling children, the senile and the feeble-minded.

This method of treatment is applicable to all fractures of the femur, including the neck, intertrochanteric, subtrochanteric, the shaft and the distal third; its use extends to unilateral fractures and injuries of the pelvis and all fractures of the tibia requiring traction. The apparatus has a utility in both operative and non-operative work, acting in the capacity of a fracture table, besides serving its main function as a reduction agent. It provides traction and immobilization for conditions of non-union, mal-union, reconstruction and bone-lengthening operations, and also for pre-operative and postoperative fixation for scoliosis.[1]

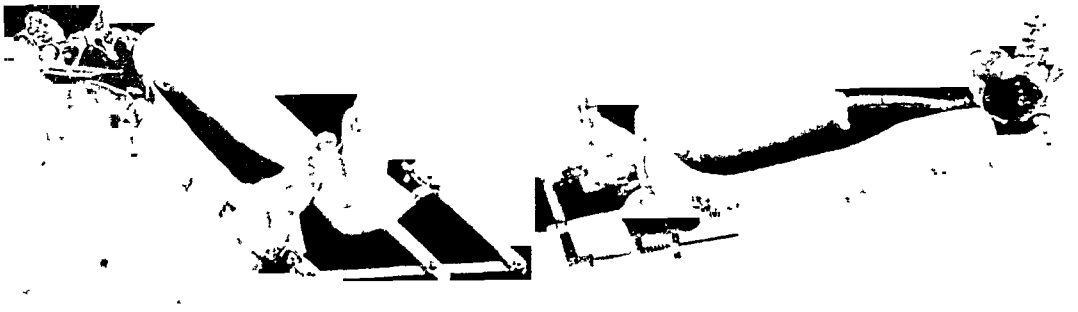


FIG. 1A. FIG. 1B.

FIG. 1. A. Fractured shaft of right femur with shortening and external rotation.
B. Reduction is accomplished and maintained even in sitting posture. Note that hip on injured side has been pulled downward by traction, forcing it into abduction, because countertraction on well side has displaced that hip upwards into adduction.

Immediate reduction, now recognized as essentially a first principle of fracture treatment, is feasible by this method; and since local anesthesia is the general order there is less danger in obtaining early reduction, and consequently the avoidance of prolonged shock, pain and undue swelling. The mechanism of the splint is simple, yet so perfected that reduction may be expeditiously carried out. Very little equipment is required besides splint, sterile set-up and plaster materials, even for the most serious case.

[1] Anderson, Roger. New method of employing skeletal traction. *Northwest Med.*, 30: 444–448, 1931.
New method for treating fractures, utilizing the well leg for countertraction. *Surg. Gynec. Obst.*, 54: 207–219, February, 1932.

* Submitted for publication August 2, 1932.

In theory and in practice the countertraction splint is the very essence of independence. The great freedom of movement allowed the patient could not be effected were the apparatus not an entity in itself, working in conjunction with no other agent than the human body. The basic feature of this method is an unalterable traction force, which operates regardless of movement of patient, and thus since the apparatus is attached to neither bed nor frame, it permits the daily use of the wheelchair. This results in economic as well as physiologic advantages: an institution needs less expensive roentgenologic equipment if the patient can be moved for the taking of films; a shortened period of hospitalization is required when the transfer home may be safely made if subsequent radiographs reveal good position. Movement is the keynote for maintaining metabolism at par and decreasing circulatory and pulmonary complications, benefits directly enhanced by the use of backrest and trapeze. The fact that countertraction is not obtained by lowering the head of the bed, as the well leg alone supplies all countertraction, accrues to these benefits. This freedom from restraint, change of position on bed or transportation by wheelchair to solarium, so increases the patient's general resistance that he feels optimistic, not only concerning the prognosis of his case, but also regarding the skill of the surgeon, a cycle continuously reacting to everyone's welfare.

APPLICATION

A hypodermic of ⅛–¼ grain morphine sulphate or without scopolamine, grain 1/100 to grain 1/300, is usually given on entrance to the hospital. A roentgenographic examination requiring two views or stereoscopic films generally precedes treatment, but the patient need not be taken from the cart on which he entered the hospital for either the films or the application of the splint. To decrease the pain and to lessen trauma the assistant should gently pull on the leg during any necessary manipulation; both legs should be washed and any wounds dressed, preparatory to reduction.

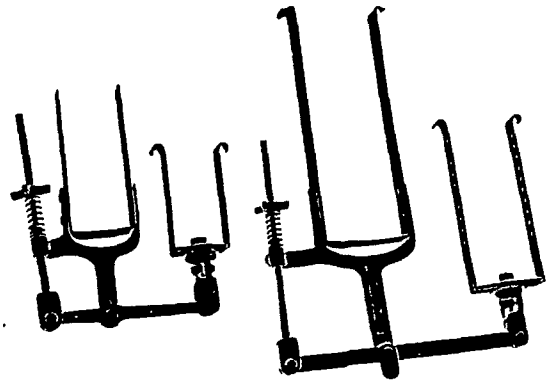


Fig. 2. Splint in infant and adult sizes.

Most of the injuries for which this method is applicable should be treated according to our *standard routine* (Fig. 3). Such a reference in the text refers to the following outline of technique:

STANDARD ROUTINE

1. Apply cast to mid-thigh on well leg.
2. Incorporate splint in cast.
3. Insert pin through tibia.
4. Apply cast to injured leg, incorporating pin.
5. Attach splint; exert traction; adjust rotation; complete reduction.

APPLICATION OF CAST TO WELL LEG

The application of this cast is of the utmost importance. Due consideration to ample protection for all bony prominences must be stressed. First, a stockinet, if at hand, is applied. The leg is well padded with sheet-wadding, especially in the region of the heel and sole; rubber bath sponge, split lengthwise in half, makes an excellent padding for the back of the heel. A thin *flexible* piece of soft felt is next applied to the sole of the foot. Then while the leg is held in adduction, the knee in complete extension, the foot at a *right angle and in slight valgus or eversion*, a plaster-of-Paris cast, extending from one inch beyond the

toe-nails to mid-thigh, is applied by using slow-setting plaster bandages, immersed the minimum time in cold salt-free water.

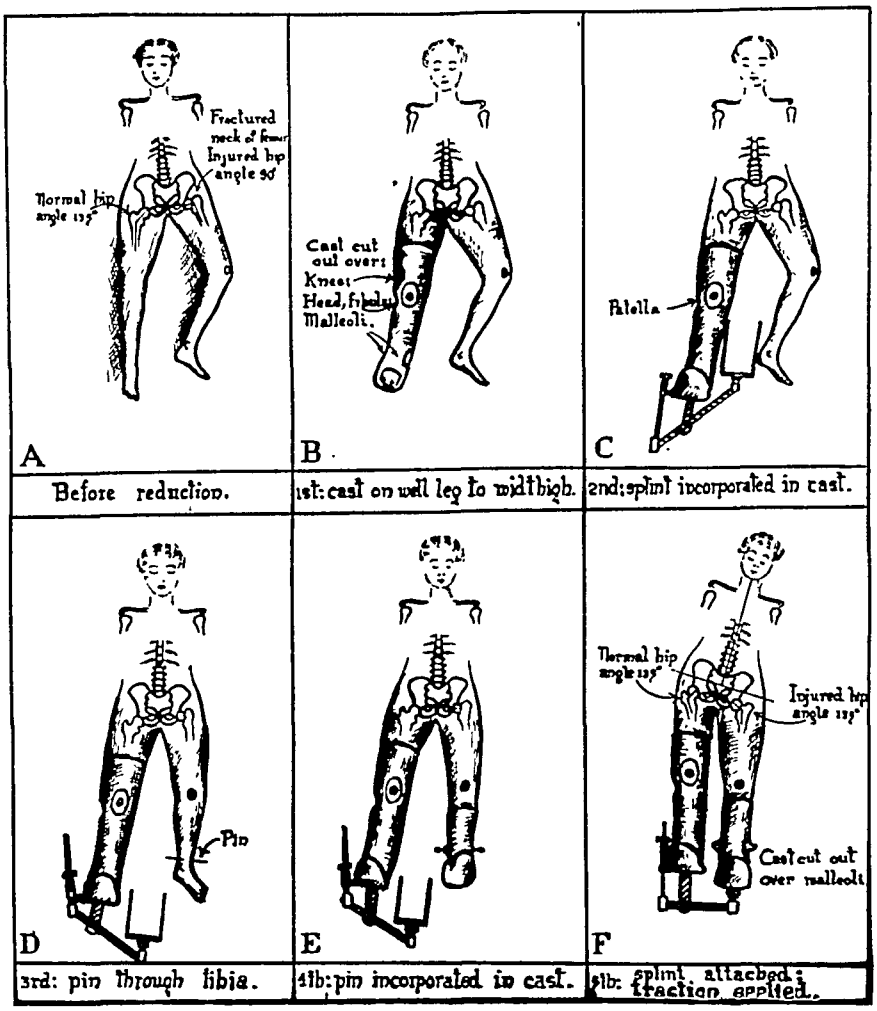


FIG. 3. Steps in application of splint, referred to in text as *standard routine.*

A thick reinforcement of the plaster, 4 inches wide, is smoothly applied to the plantar surface of the cast. Before the plaster sets the cast must be accurately molded up to fit the sole, thus insuring a balanced distribution of the countertraction pressure. The cast is made thicker around the ankle as windows of sufficient diameter are cut to expose the whole malleoli; the plaster is also cut out over the dorsum of the toes and adjacent foot and, if desired, over both sides of the little toe.

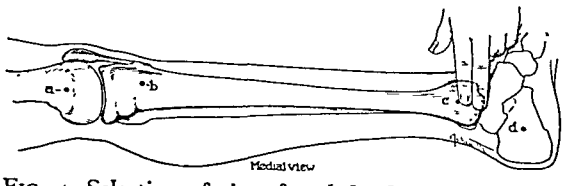


FIG. 4. Selection of sites for skeletal traction. Usual site is indicated by *c*, as employed in standard routine.

By the time these holes are cut the plaster is usually set so that the splint can be attached by an additional plaster bandage. The splint is first unbolted into its three major parts: (1) the countertraction portion, (2) the lever and screw rod and (3) the traction portion. The base of the countertraction portion (Fig. 2) is set at a right angle to the axis of the foot at a level through the center of the malleoli. The

flexible sides of the stirrup are bent by hand to fit the cast, an equal amount of space being left on each side between the stirrup and the malleoli to prevent pressure.

TECHNIQUE OF PIN INSERTION

When the skin over the fractured ends has been washed and shaved, it is scrubbed with ether and painted with iodine. Later removal of the iodine with alcohol or the use of weaker solutions eliminates the possibility of burns. The area around the ends of the fragments is anesthetized by direct injection of 20 to 40 c.c. of 2 per cent novocaine, as used by Böhler. The ankle from 6 inches above the joint to several inches below is similarly prepared, after which 2 per cent novocaine is injected into the skin and down into the periosteum on each side of the tibia. The gloves are then changed and the field draped with towels, in readiness for the insertion of the pin.

A seven-inch solid Steinmann pin of stainless steel, diameter 5/32 inch, with a four-sided, sharp-pointed end is preferred. The pin is inserted from the medial aspect straight through the center of the tibia, on a level two fingers' breadth superior to the tip of the *internal malleolus* (Fig. 4). This occasions no preliminary incision of the skin, or drilling of the bone for, if the pin is sharp, it can be forced through by rotary hand-pressure alone (Fig. 3 D). A three-inch sterile gauze sponge is spiked over the pin, down against the skin on each side, another piece of gauze about ½ by 1½ inches being twisted around the pin-ends to absorb any moisture running down the pin and contaminating the wounds. The drapes are removed and excess iodine is washed off with alcohol. Sheet-wadding, 3 inches in width, wrapped over the pin and around the leg, suffices to hold the dressing close to the skin, bandage or adhesive being entirely unnecessary. No other prophylactic against infection is required except to leave the wounds entirely alone throughout the period of immobilization.

APPLICATION OF CAST TO INJURED LEG

The cast is applied to the injured leg with the foot held at a right angle. The cast extends from one inch beyond the toenails up to a level 5 inches below the knee joint, firmly incorporating the pin (Fig. 3E). The leg is first well padded with sheet-wadding and a four-inch plaster reinforcement is applied to the sole of the foot and the cast cut out over both malleoli.



Fig. 5. These agents plus splint and plaster material are only requisites for usual fracture case.

The perforations of the traction stirrup are now connected with the pin-ends, those holes selected which permit the splint to set up most compactly against the plantar portion of the cast (Fig. 1). The traction portion is fastened to the cast by plaster bandage, the pin-ends protected by ordinary corks before being covered with plaster. Therefore, unless informed of the transfixion, the patient should be entirely unaware of the pin until its removal.



FIG. 6A. FIG. 6B. FIG. 6C.



FIG. 6D. FIG. 6E.

FIG. 6. Steps of standard routine. Patient, sixty-five years of age, has fractured neck of right femur.

A. Cast to well leg.

B. Countertraction portion of splint incorporated in cast.

C. Pin inserted through lower end of tibia.

D. Incorporation of pin in cast.

E. Traction portion of splint attached to cast of injured leg. Traction and rotation have been applied. Fracture now reduced.

After bolting together the component parts of the apparatus, the next step is to generate traction force by screwing down the traction nut (Fig. 3 F). Correct alignment, by either internal or external rotation, is obtained by operating the rotation nut (Fig. 1). *When the lever arm is drawn down to a right angle with the longitudinal axis of the legs, sufficient traction has usually been exerted.* Meanwhile both the head and feet of the patient are drawn to either the left or right side of the table, corresponding to the same side as that of the injury, while the hips are displaced toward the opposite side of the table. This position, if maintained in bed, permits the spine, paradoxical as it may seem, to set at right angles on the pelvis.



Fig. 7A.



Fig. 7B.

Fig. 7. Mrs. L. C. Comminuted fracture of left acetabulum and left ischium with central displacement of head into pelvis, non-reducible by either manipulation or by Buck's extension.

A. Before reduction.

B. After reduction with splint. Patient was removed without pain to *x*-ray room and, as this film shows, there was no disturbance of reduction.

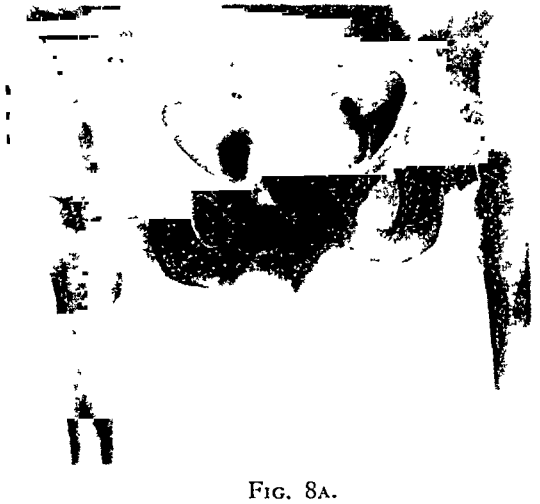


Fig. 8A.

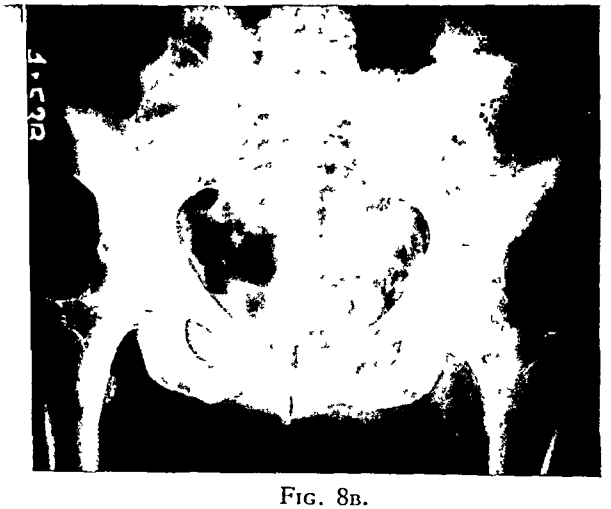


Fig. 8B.

Fig. 8. Mrs. I. LaF., aged fifty-eight years. Fractured neck of left femur, which was not detected for seven weeks and, therefore, received no treatment. Patient even tried to walk on it.

A. Before reduction.

B. Film taken day following application of splint.

At this time, or within a short while, reduction should be checked by radio-

graphs, and traction and rotation changed as indicated. A few hours after the patient is returned to his room, the cast should

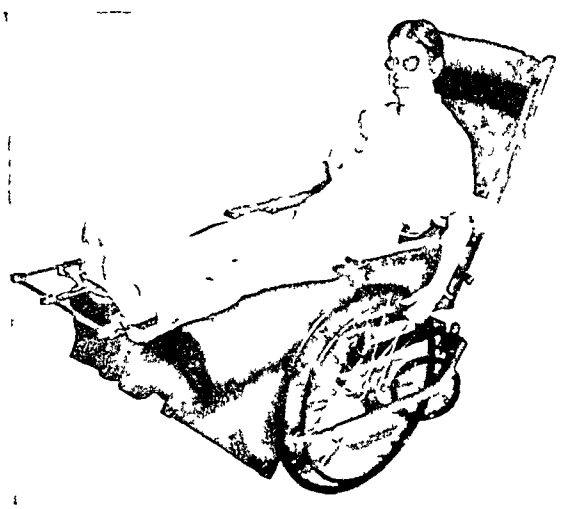


FIG. 9. Patient, with fracture of left hip, was up within a few days after application of splint.

be cut out over the posterolateral aspect of the head of the fibula and the anterior side of the knee joint.

AFTER-CARE

The patient is not only permitted to sit up but encouraged to do so and to use the trapeze freely; in a day or so he will be able to raise himself up for necessities of toilet. If the patient with a hip fracture is not in a condition of shock, the wheel-chair is often provided as early as the second or third day. The subject with heart trouble can safely be allowed to sleep in a sitting or reclining position; patients with hip and intertrochanteric fractures may be turned on the abdomen daily.

After-care is at the minimum; very little is required except perhaps to cut out over the heel, the little toe or the dorsum of the foot, or to split the upper rim of the cast. All or most of the anterior portions of both casts may be removed as conditions direct. The nurses and the patient also should be instructed to massage the knee joints and to move the knee-caps daily. It is advisable to repeat the roentgenograms as indicated

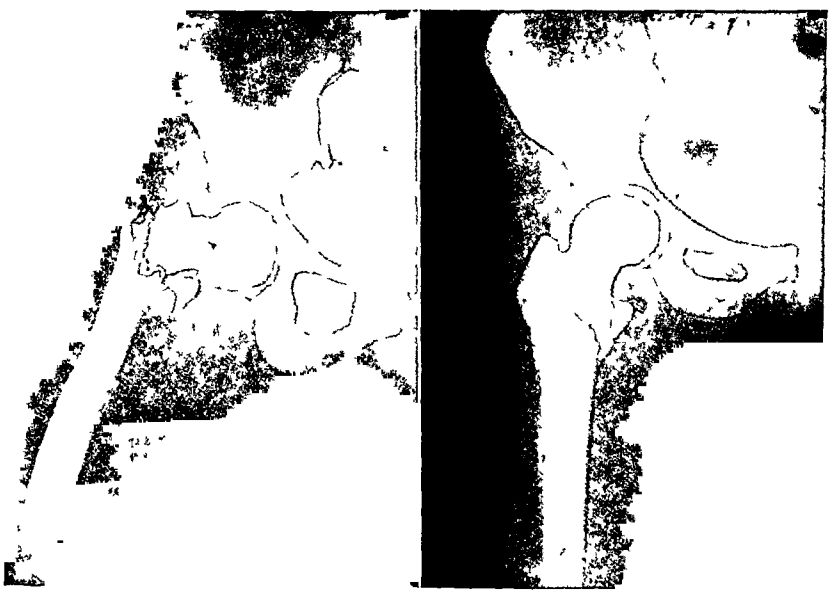


FIG 10A. FIG 10B

FIG. 10. Mrs. E. H. Comminuted right intertrochanteric fracture.

A. Before reduction.

B. *X*-rays taken immediately after application, according to *standard routine.*

If subsequent radiographs in both views or stereos verify good position, the patient may be removed home.

TIME AND MANNER OF REMOVING PIN

No attempt should be made to dress the pin-wounds from the time of the

insertion until the removal of the pin, whether this be a one-month or a four-month interval.

tures, seven to nine weeks; subtrochanteric fractures, seven to ten weeks; fractures of the femoral shaft, five to ten weeks;



FIG. 11A.



FIG. 11B.

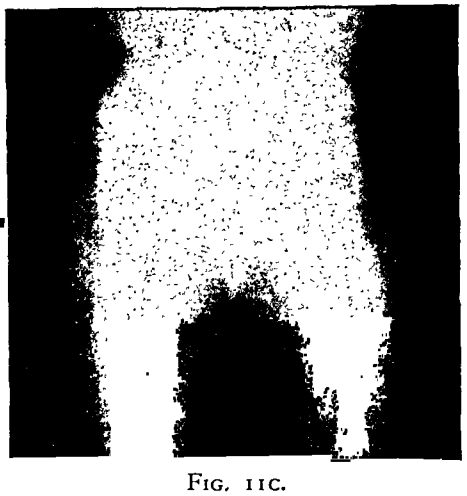


FIG. 11C.

FIG. 11. Baby B., fifteen months old. Fractured shaft of right femur; application of splint and perfect reduction obtained by Dr. L. G. Woodford.

A. Before reduction. B. After reduction. C. Final result.

The time of removal of the pin depends upon such factors as the age of the patient, the location and type of fracture and callus formation. In general, the following schedule for the period of traction is adhered to: fractures of the pelvis, four to six weeks; fractures of the neck of the femur, twelve to fourteen weeks; intertrochanteric fractures of the distal third, five to ten weeks; fractures of the tibia, four to twelve weeks; and operative cases, six to eighteen weeks.

The patient is unconscious of a securely incorporated pin; hence pain in this region is usually occasioned by looseness of the pin in the cast, a condition calling for

correction. In fact most patients are unaware of even the presence of the pin, when covered by a cast. Because of the power of suggestion, it is poor psychology to discuss this phase of treatment with patient, relatives or student nurses.



FIG. 12. Comfortable position permitted most subjects immediately following reduction, a practical application of theory that this movement in no way interferes with traction.

To remove the pin no anesthetic is required as it is usually accomplished without pain; most children are not aware of its withdrawal, if the act is screened by a pillow. To remove the pin, a hole about 5 inches in diameter is cut around the pin on the medial side of the cast, after which sheet-wadding, dressings, and crusts, if any, are removed. The diagonal ends of a 3 by 6 inch sterile gauze dressing are grasped by the fingers and twisted like a rope; after looping this over the pin, boiled tap water is applied to the center of the gauze where it contacts with the pin. With a rotary movement the plaster which adheres to the pin is removed; the same procedure is then repeated with ether, using three of these sponges. Finally, the technique is repeated with iodine. Just before the pin is withdrawn, a dressing saturated with iodine is wrapped around the pin and held close to the skin, while the pliers pull the pin through from the opposite side. A dry dressing is then applied to the wound and excess iodine removed with alcohol, but if any iodine escapes beyond the pin-wound, the cast should be further cut and the iodine removed. The pin-wounds generally need no further dressing. Unless the whole cast is removed at this time, the opposite pin-wound need not be dressed for several days.



FIG. 13A. FIG. 13B.

FIG. 13. Mrs. R. M. Comminuted fracture of shaft of right femur.

A. *X*-ray taken on receiving patient three weeks after trauma, previously treated by Buck's extension.

B. Anteroposterior view, three weeks after application of splint.

APPLICATION WITH REFERENCE TO SPECIFIC FRACTURES AND INJURIES

Fractures of the Neck of the Femur

Standard routine is employed for fractures of the femoral neck (Fig. 8), with the exception that internal rotation is increased, the amount judged by palpation of the sides of the femoral condyles. As a general rule when the proper amount of internal rotation is present, the foot will appear to be overly rotated internally. Occasionally a small sand-bag or a rolled-up bath towel placed under the trochanter will assist in rotating and elevating the greater trochanter. If desired, these patients are permitted to sit up day and

night, and within a day or so are accorded the use of the wheel-chair.

Since fractures of the neck require little

Intertrochanteric Fractures

Treatment here is also according to standard routine (Fig. 10), except that it is

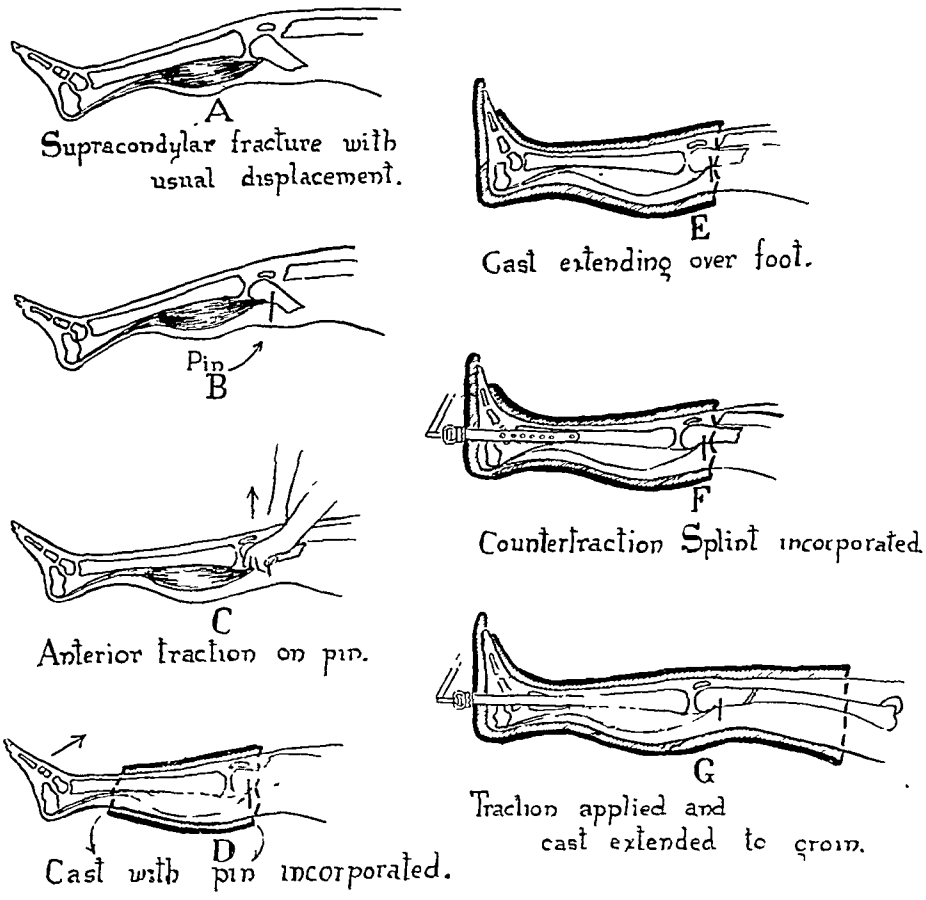


Fig. 14. Original method of treating supracondylar fractures.

traction, each week the force may be safely decreased up to that point whereby alignment is still maintained, thus insuring pressure contact between the fractured surfaces. Over-traction with its dangers, especially non-union, must be guarded against, the radiograph being the supreme arbiter in regard to retaining end-to-end contact and preserving the normal cervical angle. Stereoroentgenograms have been regarded as the desired test but Clayton Johnson's lateral technique gives additional check. If these principles are closely adhered to, better end-results with fewer cases of non-union will be attained, for this system controls these factors as does no other method.

usually best to fix the thigh in external rotation, the degree determined by study of stereoscopic or lateral roentgenograms. In the comminuted variety, especially where the lesser trochanter has been fractured off and pulled upward by the iliopsoas muscle, approximation of the fragments may necessitate a sitting position day and night in bed or wheel-chair. Some of these cases require elevation of the legs on a pile of pillows or box. This flexion assists in reduction; but perfect reduction of the lesser trochanter is unnecessary as the main objective is union of the femoral shaft, and good function can be obtained without normal reposition of all fragments.

Free use of trapeze and wheel-chair, even on the day of reduction, and shortened hospitalization are not incompatible with excellent results.

Subtrochanteric Fractures

In these fractures standard routine is deviated from, as the pin is forced through the center of the femur at a level ¼ inch above or superior to the medical condyle. Transfixion should be approximately straight through, but if the pin does not emerge at about the same level on the opposite side, it is occasionally best to withdraw and reinsert it, checking by film if necessary.

The pin is incorporated in the cast, which is extended just above the pin or to the groin, as conditions indicate. The cast on the well leg is usually extended up to the groin, since these stubborn fractures may necessitate fastening special pressure pads to this cast.[2] In many cases better apposition is accomplished by elevation of the legs on pillows or by a sitting posture.

Since the perforated sides of the traction stirrup slip over the pin when the insertion is through the lower tibia, one may seem to be confronted with an apparently too short stirrup when adapting the method to femoral transfixion. This problem has been solved by curving out the ends of the stirrup so that it is bound and fastened so securely in the plaster that it cannot be pulled out. Hence traction pull is transmitted up to the pin by the solid intervening cast.

Fractures of the Shaft of the Femur

Technique follows standard routine with the following provisos: an 8-inch pin is inserted through the femur, while the cast on the well leg is occasionally extended to the groin (Fig. 13). The splint connection between stirrup and pin is made in the manner described under subtrochanteric fractures.

If difficulty is encountered in obtaining apposition, the distal fragment can be directly controlled by cutting out the cast from around the ends of the pin in order to permit direct readjustment of the fragment by manipulation of the pin. After the desired position is attained, the pin is refastened by plaster bandage. A pressure pad, attached after exposing the fracture site through the casts, is also instrumental in effecting readjustment.[2]

Although the trapeze is permitted after the first few days, the use of the wheel-chair is dependent upon callus formation.

Fractures of the Lower Third of the Femur

For this difficult class of fractures, which include the supracondylar and intercondylar, the technique is slightly altered.[3] After-care is practically the same as for shaft fractures. The following steps in reduction are illustrated by Figure 14.

1. First, the pin is inserted through the distal end of the femur (Fig. 14B). Traction made upon the pin in an anterior direction forces the distal fragment of the femur into the same straight line with the tibia, bringing the knee into full extension (Fig. 14C).

2. The cast is applied from a level 4 inches above the ankle to 1 inch above the pin; this incorporates the pin and also holds the distal femoral fragment and the tibia in the same straight line (Fig. 14D).

3. After this segment of the cast is set, the foot is forced up to a right angle while the cast is extended slightly beyond the toenails (Fig. 14E). Holes are then cut over the malleoli.

4. The traction stirrup is incorporated in the distal end of the cast (Fig. 14F); traction is then applied and rotation corrected.

5. The cast is finally extended from the pin to the groin (Fig. 14G).

If the fracture extends T-shaped into the joint, the condylar fragments must first be aligned before the transverse fracture is reduced. Should these fragments be widely separated from each other, the

[2] The subject of a paper to be later published.

[3] Anderson, Roger. New method of treating femoral fractures in the distal third. (In press.)

medial or lateral displacement is first corrected by a specially threaded pin, the technical details being given in the author's monograph as noted above. By inserting a pair of sliding "sleeves" the fragments are forced together and held as in a vise.



Fig. 15. Reading from left to right: Mrs. N. S., aged eighty-six years, with neck of femur fracture, was turned on stomach daily; Mrs. M. S. sat up day and night because of asthma; Mrs. M. M., with fracture of right hip, made daily use of wheel-chair; Master J. H., comminuted fractures of middle and upper third of right femur. Pin was inserted through distal end of femur.

Fractures of the Pelvis

Standard routine, as previously outlined, is the procedure for pelvic fractures. Although the method is suited for unilateral fractures of the pelvis (Fig. 7), it may be used in bilateral fractures when the fractures are limited to ischium or pubic bones on the opposite side from that of the major pelvic fracture. In fact it can even be used in multiple fractures of the pelvis provided there has been no injury to that small area between the sacroiliac joint and the acetabulum on the well side. In injury or dislocation of one sacroiliac joint or in separation of the symphysis pubis, the splint has also proved successful.

After-care instructions relevant to this class of fractures include the placing of a fracture-board beneath the mattress, and a pad or baby-pillow under the small of the back for support. In most cases these patients are neither permitted to sit up nor are they turned over during the first three to six weeks.

Fractures of the Tibia

Standard routine is employed for all tibial fractures calling for continuous traction, but over-traction should be avoided since very little force is needed for this type of fracture. If a compound wound extends too near the ankle-joint, the pin is inserted through the os calcis.

For that class of tibial fractures which require traction for reduction only, provided a snug-fitting cast be applied for fixation, the splint serves in the capacity of a fracture table and is applied in the following manner. Standard routine is adopted for the well leg; the pin is inserted above the ankle or through the os calcis,

the cast on the injured leg extending just up to the fracture, incorporating the pin. The traction stirrup is then slipped over the pin and incorporated. If the ends extend above the cast they are turned back, thus permitting free adjustment of the upper fragment under traction.

The splint is then assembled and traction force exerted. Since the cast does not encompass the fracture site, the fragments may be manipulated into position and the necessary rotation effected. After satisfactory reduction is accomplished, the cast is extended up 6 inches above the knee. Traction force may be subsequently released; if roentgenographic examination shows that the fracture remains reduced, the well-leg cast and splint may be removed, leaving the traction stirrup only in the cast of the injured member. Thus the apparatus has served as an orthopedic traction table.

A modification of the well-leg method, for quick application without the use of plaster bandages, will be reported on later. For this purpose we have designed an aluminum countertraction shell, lined with rubber, to fit the sole of the adult foot and the posterior side of the leg where it is secured by straps. This device permits either wire or pin for skeletal traction.

Mal-union, Non-union, Bone-lengthening, Hip and Reconstruction Operations

Although standard technique is closely adhered to in these operations, the pin may transfix the femur, with the cast extending 2 inches above the pin. Pin, cast and splint are generally applied a day or so previous to the operation, to afford opportunity for the cast to dry and for the patient to become accustomed to this position. Before commencing to operate, a fair amount of traction force is usually generated, and the circulating nurse should be instructed how to manipulate the splint for further extension and rotation. The fact that the patient can be turned on his side or over on his abdomen for better operative exposure is a decided advantage, especially so since it in no wise interferes with traction. By removing a single bolt one may have free use of the leg for wide manipulation; the bolt can be quickly reinserted when traction and immobilization are again desired.

After the bone-graft is fastened in place and the skin sutured, in most cases the patient can be safely removed to bed. However, it may be necessary to extend the cast up to the groin over an operated femur. Under such conditions as in old, unilateral congenital hips, where a new acetabulum has been reconstructed, it may be advisable to extend the cast to the lower costal margin on the injured side in order to prevent the patient from sitting up. However, the necessary traction must be exerted before the body portion of cast is applied. Some cases call for operation through a large opening cut in the cast; the scoliotic spine responds to this type of treatment. It is clearly seen that the splint and cast apportionment conforms to whatever surgical problem presents itself.

Scoliosis, especially that of the lumbar spine, whether idiopathic or secondary to poliomyelitis, can be slowly adjusted or corrected by means of this splint. But the cast on the well leg is extended up over the body in the form of a spica cast, before any traction is applied. This corrective agent can be employed for some time before operation, which may be performed through a large opening in the cast. On the other hand, fusion operation may be performed first. The splint is later applied according to standard routine with the extension of the cast upwards to the mid-dorsal region, after which traction, the corrective force, is exerted.

ADAPTATION AND SIZE OF SPLINT

The splint has been so constructed that it works equally well with other forms of skeletal traction than the pin. Moreover, the pin, wire or tongs need or need not be incorporated in the cast; for the latter the splint is equipped with an adjustable

foot-plate. Should there be a contraindication to skeletal traction, Buck's adhesive can be used equally well with this countertraction.

This method which has been successfully employed for subjects ranging from a fifteen-month old infant to a three-hundred-pound man demonstrates its wide adaptability, the apparatus being made in adult, youth and infant sizes.

SUBSEQUENT CARE

Continued complaint is a definite indication of something wrong. This can generally be attributed to faulty application of the cast. Permanent indentations in the cast with their inevitable pressure sores will result if the assistant supports the weight of the leg with the fingers instead of the palm. But before the plaster sets the hand must be frequently moved in its position against the leg. Pain in the region of the knees can be avoided by holding the legs fully extended during the application of the casts. Pressure is corrected by splitting or cutting away the offending portion of the cast. In fractures of the tibia and some femoral shaft fractures it is advisable to split the cast completely.

Complaint of pain in the well hip is usually attributable to over-traction, so easily obtained with this method. In spiral fractures, especially, the error of over-traction occurs because one is so liable to misinterpret overriding for what is complete reduction. We must remember that, except for fractures of the femoral shaft, surprisingly little traction is necessary for reduction and immobilization. However, the exigencies of the case may necessitate such an unusual amount of traction as to be the source of pain in the region of the well hip or trochanter. This generally disappears in a short while, though its severity may demand a temporary release of traction, which later can be steadily increased as the force is tolerated. On the other hand, if the films do not reveal over-traction, sedatives or hypodermics may be indicated for some cases. Maximum traction is only required, as a rule, during the first week and can be reduced during the ensuing period. In this sequence of technique the radiograph will be the judge. In the final analysis the best results are attained with a contented patient, and immunity from pain is naturally inferred.

The usual precaution against the danger of too early weight-bearing must be observed. This warning is especially relevant to fractures of the femur; with many shaft fractures it is advisable to keep the patient on crutches or a walking-caliper for a period of six months from the date of accident, while those of the neck frequently require weight protection for a year. After the cast and splint are removed the leg should be closely watched as muscle tension on soft callus results in bowing. In this event it is advisable to apply some form of temporary traction or to resort to corrective cast or brace equipped with pressure pads as necessary.

PARTICULARS FOR CARE OF APPARATUS

Use does not impair the value of the splint. It will not rust and it can be polished with ordinary nickel polish, or a machinist can quickly buff it up.

It is not advisable to boil the splint because aluminum darkens under such procedure.

When the cast and splint are removed, only water should be used to soften the plaster as other solvents act on aluminum.

The same pin may be repeatedly used if the end and edges are resharpened, but the point should be retempered if sharpened on the emery wheel.

By the removal of just two bolts the splint can be divided into three parts—countertraction, lever and rod, and traction—thus facilitating attachment of splint to casts.

The exposed parts of the splint may be wrapped with sheet-wadding to keep the threads, connections and lever arms free from plaster while being incorporated in the cast.

IMPORTANT TECHNICAL CONSIDERATIONS

1. Hold the well leg in adduction at the hip while applying the cast.

2. Hold the well foot at right angles with the foot in slight valgus (eversion or pronation of the foot).

3. Pad the sole of the well foot with *thin, soft, flexible felt* and with a goodly amount of sheet-wadding. A four-inch reinforcement of plaster should be smoothly applied over the entire plantar surface of the cast on the well foot; this should be moulded accurately to fit the sole.

4. Cut sufficiently large openings in the cast over the malleoli of both injured and uninjured ankles before incorporating the splint.

5. The cast should be cut out later over the posterolateral aspect of the head or heads of the fibulae.

6. Pain or trouble with the well leg is usually attributable to careless application of the cast. Continued complaint demands a change of cast. Pain in the region of the well hip is usually indicative of too rapid or too much traction. In most cases when the lever arm is drawn down at a right angle with the longitudinal axis of the leg, the requisite amount of traction has usually been exerted.

7. *Avoid over-traction*, especially in fractures of the femoral neck, by checking with repeated roentgenograms. *Over-traction is a frequent cause of non-union.*

8. Remember the warning of the Cooperative Committee on Fractures: "It is the doctor behind the splint and not the splint that counts in the treatment of fractures."

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*Continued from p. 122.

CHOLESTEATOMA ORIGINATING IN THE SKULL BONES

CAUSING SYMPTOMS OF INTRACRANIAL PRESSURE*

BERNARD J. ALPERS, M.D., AND REED HARROW, M.D.

PHILADELPHIA

INTRODUCTION

CHOLESTEATOMATA originating in the bones of the skull are found chiefly in two situations: in the temporal bone, and in the other bones constituting the cranial cavity. Of these two types the cholesteatomata found in the temporal bone do not concern us. In this situation they develop following chronic middle ear disease, with occasional extension into the cranial cavity. They do not represent primary tumors, but are rather secondary to the chronic inflammations of the middle ear. As such their structure differs from the primary cholesteatomata in the total absence of an epithelial envelope, as well as of the characteristic plant-like cells which make up the tumor. These tumors are filled with lymphocytes and leucocytes and squamae in all stages of fatty and granular changes.

On the other hand, in very rare instances, there are found cholesteatomata which take their origin in the bones of the skull, remote from the temporal bones and middle ear. Such tumors have the typical structure of a cholesteatoma. They possess a true epithelial envelope, and contain the typical flat cholesteatomatous cells. They produce a characteristic *x*-ray picture which it is important to recognize, and may or may not cause symptoms of cerebral compression.

It is such a primary cholesteatoma originating in the occipital bone which we wish to report.

CASE REPORT

J. J., Surg. No. 18752. Impairment of vision, headache, nausea and vomiting, and bony defect in right occipital region of twelve years' duration (1918–1930). Emotional unbalance, reduced visual acuity, binasal quadranopsia, secondary optic atrophy. Large bony defect shown in right occipital bone by *x*-ray. Biopsy revealed cholesteatoma. Evacuation of cyst, entirely extradural. Uneventful recovery.

History: On April 24, 1930, J. J., a white woman of fifty-two, was admitted to the Neurosurgical Service of Doctor Charles H. Frazier, complaining of failing vision in the right eye, headaches and nervousness. She dates the onset of her illness back to 1918, at which time she noted a small "soft spot" in the right occipital region. There were no symptoms referable to this particular site at that time, but the patient noticed that the vision of the right eye was becoming impaired, and that there was blurring after reading for any considerable time. The visual impairment persisted. Two years later (1920), she developed so-called "dizzy spells," described as beginning with a feeling of alternate heat and cold over the entire body, a desire to relax all muscles, and nausea followed by the vomiting of bile-stained fluid. She complained also of generalized headache, more severe in the occipital region, and a peculiar feeling in the head, at times resembling "moving wheels," aggravated by a sudden change of position. After the attack, she felt completely exhausted and drowsy. During the intervals she felt fairly well.

The patient was studied at a local hospital at this time, but according to her statement, no diagnosis was made and she was discharged unimproved.

She suffered from frequent attacks, similar to those described, during the succeeding four years (1920–1924), after which there was an apparent abatement. At no time was there any disturbance of gait, or any incoordination of the upper or lower extremities. The vision of the right eye became progressively impaired, with increased blurring.

* From the Neurosurgical Wards and Laboratory of Doctor C. H. Frazier in the Hospital of the University of Pennsylvania, Philadelphia. Submitted for publication December 23, 1931.

In 1925, the patient developed pulmonary tuberculosis with a reported positive sputum. She spent a year at a mountain retreat and made a remarkable recovery, resulting in the gaining of 95 pounds, and in a complete subsidence of symptoms. The menopause appeared this same year, adding to the general nervousness of the patient.

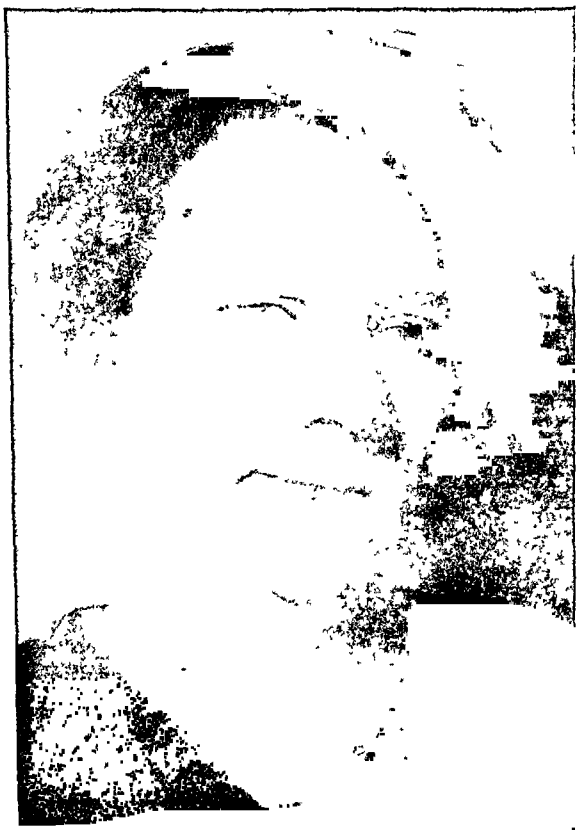


Fig. 1. Postoperative photograph of patient after complete removal of a diploic cholesteatoma in right occipital bone.

It was not until two years ago (1928) that the patient noted that the headaches in the right occipital region were becoming severe, and that there was tenderness at the site of the bony defect. The "soft spot" which she had first noticed twelve years before, had increased slightly in size until it now admitted the end of a finger. Since 1924 she had been comparatively free of dizziness, nausea and vomiting. The vision of the right eye became neither better nor worse, and although it was considerably less acute than that of the left, she was able to read with the aid of glasses. Blurring still occurred after protracted reading. These symptoms persisted to the time of admission.

On admission, the patient complained of impaired vision in the right eye, a return of "dizziness," nausea and vomiting, and three different types of headache: a dull throbbing ache at the site of the bony defect in the right occipital region, a pain over the vertex, burning in character; and a generalized headache that accompanied the "dizzy spells." She was greatly upset over her condition, showing a marked degree of depression at times, crying for long periods and fearing that she was becoming insane.



Fig. 2. Large area of rarefaction in right occipital region. In center of this area of rarefaction there is an area of increased density probably due to sequestrum.

For the first few days after admission to the hospital, she appeared to be very depressed, reticent and refused to talk about her trouble, stating that she was "tired of being questioned by doctors." On the third night, the resident physician was called to her room where he found her lying in bed, threshing about, holding her head in her hands, staring wildly and crying loudly that she was dying, that there was a machine in her head that was driving her insane. She complained bitterly of headache and nausea, vomiting small amounts of gastric contents. It was necessary to administer a hypodermic to quiet her. During the next few days frequent similar attacks occurred

and following each one, she remained in bed throughout the day. She was often childish in her attitude and was said to be facetious.

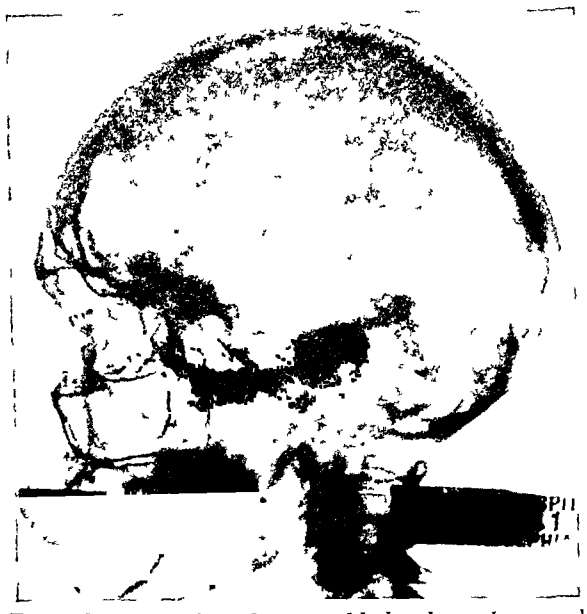


FIG. 3. Large portion of inner table has been destroyed and there is some expansion of diploic region.

In the past medical history there was revealed an attack of typhoid in 1910, a history of gallstone colic and cholecystotomy in 1916, and influenza in 1918.

A history of cranial trauma, not particularly clear-cut, but nevertheless present, was obtained. In 1912, while standing on the steps of her home, she was hurled violently across the street following an explosion occasioned by the search for a leaking gas pipe with a lighted match. She was certain that she struck her head in the fall, but suffered no change in consciousness as a result.

Examination: Physical examination revealed a head of normal contour. In the right occipital region was a slightly tender "soft spot," about the size of a quarter. From palpation alone, it was quite evident that there was complete bony destruction of the underlying skull at this site. No pulsation was noted. Several small xanthomata appeared on the upper lids of both eyes. Examination of the chest showed impairment of percussion note over the upper left lobe, with diminished breath sounds and slight limitation of expansion. No râles were heard. The lesion was probably the scar resulting from the old tuberculous infection. The heart was essentially negative. Blood pressure, 166/98. The abdomen was obese and showed an incisional scar in the upper right

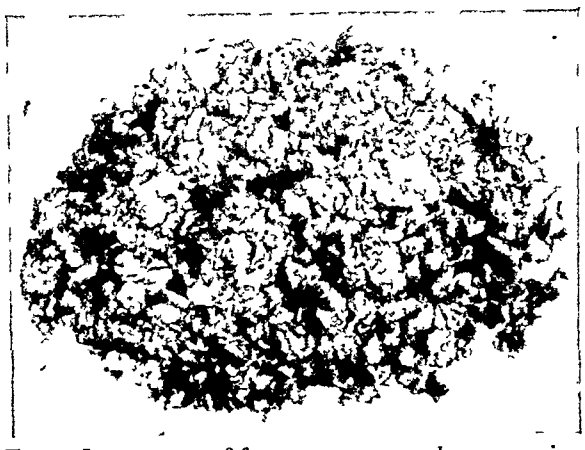


FIG. 4. Large mass of fragments removed at operation, together with capsule of tumor.

quadrant. No organs or masses were palpable. The extremities were entirely negative.

The patient was fully oriented, fairly cooperative, and attentive. The emotional state was variable, at times a marked mental depression and apathy being present, at others an emotional unbalance, with hysterical manifestations. The effects of a long period of introspection, anxiety and concern over her physical condition were well marked. The station and gait were normal. There were no motor or sensory abnormalities noted. The pupils were round and regular, the right measuring 2–5 mm., the left 3 mm. Reaction to direct and consensual light, accommodation and convergence were normal. Ocular movements were complete in all directions. Visual acuity: O.D. 6/60; O.S. 6/20. Ophthalmoscopic examination showed the retinal arteries contracted, the veins tortuous but normal in size, the disc margins irregular and gray in color, the physiological cups filled and the appearance of secondary optic atrophy on both sides. The visual fields showed a lower binasal quadrant cutting with slight concentric contraction. There were no visual hallucinations.

Lumbar puncture showed the cerebrospinal fluid to be under a pressure of 150 mm. of water. The cell and protein content were within normal limits and the Wassermann and colloidal gold tests were negative. The blood Wassermann reaction was negative. The urine

showed a trace of albumin and an occasional hyalin cast.

The *x*-ray of the head showed a large bony defect in the occipital region which occupied most of the occipital bone on the right side. This defect appeared to be similar to that seen after a craniotomy, except that it was not quite as regular. In its midst was a large longitudinal strip which was denser than the surrounding absorbed portion, and was presumed to be a sequestrum. Surrounding this, and occupying practically the entire bony defect was a large area of rarefaction and decreased bone density. The edges of the bony defect were irregular, with spur-like projections extending out into the area of decreased density. The edges were rounded and smoothed off except for the spurs already mentioned. Lateral stereoscopic views showed a large cavity which corresponded to the area of decreased density. The outer table of the skull was thinned in some places. The inner table had been bellied out and formed the inner wall of the rarefied central area. The inner table did not form a continuous line, but was interrupted for rather long stretches, due presumably to its destruction in localized areas. In the lateral views could be seen fine strands of denser tissue which ran across the absorbed area, and gave it a somewhat reticulated appearance. Bone destruction was rampant everywhere.

Because the exact nature of the lesion was uncertain, a biopsy was made on May 6, 1930, by Dr. Frazier. A button of bone was removed from the right occipital region in the neighborhood of the cranial defect, and beneath the bone "there was a circumscribed mass of soft, putty-like tissue which suggested the degeneration of a cyst." Histological examination showed the specimen to be the contents of a cholesteatomatous cyst.

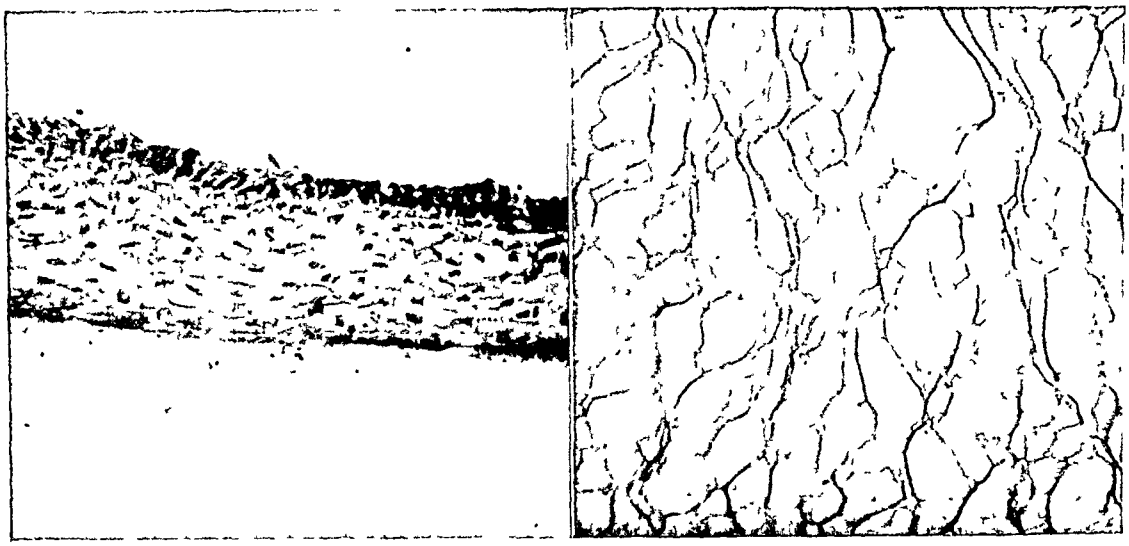


Fig. 5A. Fig. 5B.

Fig. 5. A. High power view showing stratum granulosum with typical flat, polygonal cells characteristic of this layer. Stratum durum not shown. B. In this view are seen characteristic cells of stratum granulosum. There are some features here too of stratum fibrosum which lies immediately above this layer.

Operation: On May 16, 1930, Doctor Frazier evacuated the cyst, about 2 ounces of the above described material being obtained. "There remained lining the cavity what appeared to be the wall of the cyst which could be removed piecemeal by peeling it off. Each section looked somewhat like the skin of an onion." The cavity lay behind the occipital bone and was unilateral for the most part, although it seemed to extend somewhat across the midline. It was more or less the shape of a wedge with its base toward the surface. The cyst was entirely extradural. No attempt was made to repair the cranial defect at this time. A small drainage tube was inserted into the cavity and the wound closed by buried catgut and superficial silk sutures.

For the first few days following operation, the patient complained of similar attacks of dizziness, and there was no appreciable improvement in her mental attitude. However, by the time of her discharge from the hospital,

June 18, 1930, she stated that she felt considerably better, the headaches especially being much less severe. From the time of operation, the wound continued to drain a serous discharge, but the cavity had continued to decrease in size until its volume was measured at 1 c.c.

Shortly after discharge from the hospital, the cavity became infected and a chronic draining sinus resulted. On August 21, 1930, she was again admitted to the hospital and the wound reopened, curetted and thoroughly cleaned. Examination at this time showed the patient's only complaint to be the draining sinus at the operative site. The "dizzy" spells and headache had completely disappeared. She was much brighter and more cheerful. Visual acuity was thought by the patient to be improved, but showed no objective changes.

Pathological Examination: The tumor had the typical mother-of-pearl appearance of these growths. It was extremely friable and fragile, and its layers could be peeled off readily. Among the characteristic large plant-like cells were numerous cholesterin crystals. Although the tumor was removed piecemeal much of the capsule was removed with it, and examination of this revealed an outer homogeneous layer of great thickness, under which lay flattened cells four or five layers in thickness. Since special fixatives were not employed, it is not possible to state whether keratohyalin granules were present in the cells of this layer which Bailey[1] has termed the stratum granulosum. Microscopic studies failed to throw any light on the relation of the tumor to the cranial bones. Nowhere in the tumor could bone be demonstrated, and the tumor capsule itself peeled away readily from the bony surroundings. There was no invasion of the latter.

CLINICAL FEATURES OF THE CASE

There are several points of interest raised by our case: the characteristic *x*-ray findings which should make one suspicious of a diploic cholesteatoma, the relation of the mass to the trauma sustained about six years prior to the onset of symptoms, and the complete subjective improvement following the operation.

The relationship of trauma to the onset of symptoms or to the development of these tumors is, of course, a difficult one. The structure of the cholesteatomata found in the cranial bones is in all respects typical of a primary tumor of this sort. Were the trauma responsible for the development of the tumor we should expect a so-called granulomatous type of cholesteatomatous tumor such as is seen in the temporal bone subsequent to prolonged middle ear infections. There is nothing to suggest this structure in these tumors, and we must therefore not look upon the trauma as the cause of the tumor *per se*, whatever else its rôle may be in the clinical picture. There is further the striking fact that these tumors may develop in the bones of the skull without a history of trauma of any degree. It is nevertheless of more than passing interest to call attention to the history of trauma obtained in the few cases of this sort which have been encountered by others. In Weinlechner's[2] case, a man of forty-five had received a blow on the head when eight years of age, followed by a swelling of the head, and followed four years later by loss of consciousness. Blecher's[3] patient fell on his head and developed a persistent swelling in his parietal region following the trauma. The trauma in Borchardt's[4] patient consisted of a fall on the head from a distance of 4–5 meters followed by subjective symptoms which were referable to the fall. Cushing's[5] patient fell from a horse and struck his head, but suffered no loss of consciousness. Four months later he developed a depression in his temporoparietal region. What the relation of the traumata to the subsequent development of these cases has been is hard to say. No one in our present state of knowledge concerning the origin of cholesteatomata would venture to say that the tumors had developed as a result of the injury. No one has demonstrated that trauma may produce a cholesteatoma. We are justified in saying, however, that following trauma some of these cases of diploic cholesteatomata have

developed persisting swellings at the site of injury, swellings which have later proved to be cholesteatomata.

The postoperative recovery in our case was complete from the subjective standpoint. The mental symptoms, headache and dizziness disappeared entirely following the operation. The visual field defect persisted however, despite the fact that the tumor was entirely extradural and was removed completely, and despite the fact also that there was no evidence of a recurrence. The tumor was removed piecemeal because its nature was not identified until after the bony defect had been entered at operation. It was possible nevertheless to remove the capsule, though it would have been preferable to have removed tumor and diseased bone intact in one large flap. Despite the failure to do so the operative result was extremely satisfactory.

CASES OF CHOLESTEATOMA IN THE CRANIAL BONES

The story of the cholesteatomata has been told many times. Cruveilhier[6] in 1829 first named them pearly tumors because of their striking mother-of-pearl appearance. They were christened cholesteatomata by Johannes Müller[7] in 1838, and renamed pearly tumors by Virchow[8] in 1855. Since then the terminology of these tumors has been a subject of rather heated controversy, some objecting to the noncommittal pearly tumor, and others rejecting cholesteatomata as a name for these tumors because of the presence of cholesterin crystals in many other tumors not related to this group and their absence in some tumors called cholesteatomata. And there the matter stands at the present time. It seems to us that the term cholesteatoma is at least well accepted and established through common usage, and should therefore be used in preference to pearly tumors, even though the objections to the term are well-founded.

There have been very few cases of cholesteatomata arising within the bones of the skull. The condition is rare. In Dr. Frazier's series of brain tumors cholesteatomata represent one-third of one per cent of all intracranial tumors exclusive of pituitary tumors, and among these the variety which is found primarily within the bones of the skull is rare. Probably not more than a dozen cases have been recorded in medical literature.

The location is not constant. The tumors are found in the frontal bone (Esmarch, Weinlechner, Wotruba, Blecher), in the temporal bone (Virchow), in the occipital bone (Borchardt, Alpers and Harrow), in the parietal-temporal area (Cushing), and in the temporo-occipital region (Koerner).

The symptoms which result from these tumors also vary. The tumors are slow growing, remaining extradural throughout their course, and they therefore tend to produce very few evidences of intracranial disturbances. Sometimes their presence is heralded only by a bony swelling or defect. Often the only evidences of their presence are found in the vaguest sorts of symptoms such as headaches, dizziness and vague mental symptoms. Most rarely, as in our case, signs of intracranial disturbance are produced, due possibly to the compression of the brain by the bowed-out inner table of the skull.

In the last analysis the diagnosis of a cholesteatoma within the bones of the skull depends on the *x*-ray findings. The clinical symptoms and signs are usually so vague as to make an accurate localization almost impossible. The presence of a bony swelling or depression which is visible grossly and seen in greater detail in the *x*-ray pictures establishes the diagnosis as well as the localization. The tumor appears to originate between the two tables of the skull. Cushing states that the tables become separated by the tumor, are thinned by pressure and finally absorbed, the inner table as a rule suffering more than the outer. There is a bowing-out of both tables of the skull (Blecher), bone absorption taking place chiefly within the substance of the bone. Occasionally there is erosion

of the inner table of the skull by the tumor. This may occur in one or more places, but it does not seem to reach a size which permits the tumor to break through. The cerebral symptoms which these tumors produce are not the result of extension of the tumor through the bone and into the cranium, so much as to the compression of the brain by the arching of the inner table of the skull.

The *x*-ray picture produced by one of these diploic cholesteatomata resembles, as Cushing has so aptly said, the sort of defect which might have produced by an old craniotomy. Since the early cases reported were not studied from an *x*-ray standpoint, it is only recently that the roentgenologic features of these tumors have become known.

The cholesteatomata arising in the bones of the skull are very slowly growing tumors. Blecher believes that they are usually noticed first at ten or fifteen years of age. They destroy the cranial bones in which they grow, but are only locally malignant. While they erode the inner table of the skull, they do not penetrate through the dura and invade the brain substance. Both by virtue of their slow growth and their strictly local nature there tumors are benign and can be successfully removed surgically. The art in their removal consists in the complete extirpation of the epidural membrane which surrounds them. So long as this is not removed the possibility of a recurrence is imminent. Furthermore the membrane can be removed most completely before the cyst is emptied of its contents. In our case it was felt that the entire membrane had been removed despite the fact that the tumor was removed in fragments and the contents emptied before the removal of the tumor. Thus far no recurrence has been noted.

CONCLUSIONS

1. A rare type of cholesteatoma, primary in the occipital bone, is reported.
2. The tumor was successfully removed at operation.

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TREATMENT OF TRAUMATIC TETANUS

WITH REPORT OF FIVE CASES*

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GREENVILLE, S. C.

BEFORE going into the treatment of tetanus, I wish to mention some of the important facts about this condition. There are four diseases which are very similar: shingles, rabies, tetanus and acute poliomyelitis. The toxins of these diseases are alike in their selective influence upon the nerve tissue. The mortality rate of tetanus is variously given. In some of the most recent statistics the rate has been placed at 85 per cent after convulsions have begun, and 95 per cent where convulsions begin within ten days from the time of the injury. The mortality rate will, of course, be lowered when the symptoms come on after ten days and when the wound is on the extremities.

The cause of death in tetanus is exhaustion or cardiac failure due to long-continued or severe clonic convulsions or respiratory failure due to spasm of the glottis or diaphragm. Calvin and Goldburg,[2] Chicago, state that the mortality from tetanus at the Cook County Hospital has not decreased in the past fifteen years in spite of the fact that in the last five years larger amounts of antitoxin were used and it was given intraspinally more often. A larger number of cases occurred between the ages of ten and fifteen years. Tetanus was five times as common in males. Tetanus following gunshot wounds had a high mortality rate of 95 per cent. The incubation period in 56 per cent was under ten days. The mortality at Cook County Hospital of cases under ten days was found to be 84 per cent.

The treatment of tetanus is difficult because it is not known the patient has tetanus until it has reached his spinal cord and manifested itself by muscular spasm. It is for this reason that a planned vigorous treatment must be carried out in all cases early; do not defer more active measures until the symptoms become more severe. Every case of tetanus is serious enough to receive the most vigorous treatment and be prepared to institute it at once. We are not treating a disease which we treat symptomatically.

The treatment resolves itself into (1) neutralization of the toxins present; (2) relaxation of muscular spasm as near as possible; (3) prevention of absorption of toxin.

1. NEUTRALIZATION OF TOXIN

In my opinion the toxin already found should be met with a sufficient amount of antitoxin intraspinally and intramuscularly, but not intravenously. My reason for not giving it intravenously is because of the shock and prostration to an already weak and tired patient due to anaphylaxis. During the war no case of tetanus in France recovered if treated by the intravenous route only. The intraspinal administration is less often followed by anaphylaxis. All patients should be tested for anaphylaxis and if there is any reaction they should be desensitized. This can be accomplished by this simple method. (1) Take 5 c.c. of serum, dilute with 50 c.c. of normal saline, inject 1 c.c. of the mixture intravenously; four or five minutes later inject 3 c.c.; five minutes later 10 c.c. are given and then 25 c.c. of the mixture can be given. After a further delay of fifteen minutes the serum can be given intraspinally or intramuscularly. Another point to be remembered about the administration of serum is that it may be forty-eight hours before all the serum is absorbed when given subcutaneously, whereas when given intramuscularly it is absorbed within twelve hours. While I am discussing antitoxin there is one thing that should be mentioned concerning its use in the prevention of tetanus. The

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French found that the period of immunity produced by antitoxin lessened with each succeeding dose. After the first immunizing dose they found it to be good for twenty-one days; after the second dose, seven or eight days; and after a third dose, even a shorter time. Therefore in an old case of gunshot wound, they gave the patient 15,000 units two days before performing a secondary operation lest the operation activate some dormant bacilli. The amount of antitoxin I have used in this series of cases has been 7500 units concentrated intraspinally, 40,000 subcutaneously, or better still, intramuscularly, this serum all to be given the day I see the patient. I have not used larger doses because I felt these were sufficient. I have not repeated my antitoxin because I think that to give many large doses over a considerable period invites the anaphylaxis reaction. When I began the series, it was at the suggestion of my partner, Dr. C. B. Earle, that I begin the combined use of carbolic acid and serum. I have found since that the French have used the combination of serotherapy and carbolic acid. In what manner the carbolic acid acts, I do not know any more than the physician knew how quinine acted before he knew the cause of malaria. I am convinced that carbolic acid has a curative action on tetanus if the patient is treated early enough to get the effect of the treatment. The method of treatment is to inject a 2 or 3 per cent solution of carbolic acid in the amount of 1 to 5 grams in twenty-four hours, preferably to be given twice a day, the first dose to be injected around the wound, and if it is on the extremity, to inject up along in the region of the nerve trunks. The dosage of carbolic can be increased daily the urine being watched twice daily for a smoky cloud. As soon as the urine becomes dark and smoky in appearance, stop the carbolic acid.

2. Relaxation of Muscle Spasm

Protecting the patient from external stimuli needs consideration; that grand jumble of being admitted to the hospital should be smoothed out a bit. There is no danger of tetanus to the other patients, but the other patients are a great danger to the patient with tetanus. He should have a quiet room and if possible a quiet nurse.

I have not used morphia in this series of cases to control the spasm, because no drug will control the spasm completely and large quantities of morphine certainly will not help the patient. I have used sodium amytal for these cases and find it has worked well toward relieving the patients of that horrible anxiety they all have. The patients have all been conscious the entire time during their illness, I kept them under the influence of sodium amytal for weeks without any bad effects on the kidneys, and with no excitable or delirious effects.

3. Prevention of Absorption of Toxin from Wound

This was accomplished by the application of pure carbolic acid in the wound if it had not healed, and the injection of a 2 per cent carbolic solution around the wound. I do not believe it is wise to be continuously probing or treating the wound because excitement of some dormant bacilli by such practice may occur. The fluid balance of the body must be kept up. This can be done by proctoclysis of soda saline solution, or intravenous glucose 10 per cent, when the patient has much trouble swallowing.

Polloni[3] gives a survey in April of this year of a twelve-year period in Rome. Eliminating the fulminating cases, he had 53 cases treated with carbolic acid alone with a mortality rate of 30 per cent.

Case Reports

Case 1. G. O., Chart No. 2491, City Hospital. Referred by Dr. H., boy, aged fifteen. Admitted to hospital November 26, 1929.

History of Injury: Cut his hand with rusty axe which had been lying in the yard. Treatment of wound consisted of washing the hand and then putting iodine in wound. The wound was dressed by a member of the family until

it healed. This injury occurred three weeks before patient was admitted to hospital. Three days before admittance to hospital patient complained of cramps in the neck, back and shoulders; also had some pain on swallowing. Dr. H. was called and patient was sent to hospital November 26, 1929.

Examination at hospital revealed a boy fifteen years old, under-nourished, anemic, unable to visualize, throat, a spastic smile (*risus sardonicus*), opisthotonos, marked rigidity of abdominal muscles, exaggerated knee jerk, perfectly conscious, but talked with some difficulty due to inability to use muscles of mouth. Temperature on admittance 101°F., pulse 136, respirations 34, blood count 9950.

Course: On the following day this picture changed considerably; there were definite convulsions with marked general muscular spasm, voiding involuntarily, very restless, least noise caused convulsions. Patient's chief complaint was pain in the back. A very sick and restless patient.

On November 29, temperature, pulse and respiration began to come down, but patient developed pain in chest and cough. Still unable to take much fluid.

December 3, temperature, pulse and respiration about normal. Muscular spasm and pain about the same.

December 7, up in chair, still has spasm in muscles of back, on a soft diet. Discharged December 16, after twenty-one days in hospital; patient can walk with the aid of someone helping him.

Still muscle spasm in leg and back.

Examination in office a month later all muscle spasm gone.

Case II. M. H. File No. 1977, 1930. Referred by Dr. D. Girl, aged nine years. Admitted to hospital September 1, 1930.

History of Injury: Patient stuck splinter in foot August 27, in chicken yard, just four days before being admitted to hospital. Wound was treated by mother who was a very sensible woman and a graduate nurse. Wound healing nicely. On August 31, ate breakfast with difficulty. September 1, patient still complaining of inability to open mouth quickly; had pain in neck twice that day. Dr. D. called, patient brought to hospital on night of September 1, walked into hospital; was able to open mouth, but was very slow in doing it.

Examination showed no exaggeration of reflexes, no great amounts of stiffness of neck muscles. Patient was put to bed and no treatment given (a mistake). Temperature 99°F., pulse 88, respiration 20.

Course: At 6 A.M. patient had slight convulsion followed by pain in neck and back. Unable to open mouth. Blood count 11,700, polymorphonuclears 73 per cent. Urine negative, marked opisthotonos, spastic smile, light phobia, marked exaggerated knee jerk, a very restless and uncomfortable child.

Treatment was begun four hours later than it should have been. September 3, temperature 102°F., pulse 90, respiration 38. Fluids given by proctoclysis, 5 ounces every four hours. Patient in spasm most of time, spasm lasting for two minutes.

September 5, pain in chest, spasm lasting one minute. Unable to get anything but milk and water between teeth.

September 7, pain in chest and back, convulsion lasting about one minute, unable to open mouth.

September 8, temperature 102°F., pulse 108, respiration 28. Pain in back and chest. Still very rigid spine.

September 10, temperature 103°F., pulse 116, respiration 26.

September 12, temperature 100.4°F., pulse 108, respiration 30. Carbolic acid discontinued because of deep, smoky urine.

September 15, temperature, pulse, respiration about normal. Patient much more comfortable and up in chair.

September 16, patient comfortable but still unable to bend her back or open mouth ½ inch. Temperature 98°F., pulse 80, resp. 20.

Patient discharged on the sixteenth hospital day to go home. Patient still not able to walk.

A month later office examination failed to reveal any evidence of muscle spasm.

Case III. R. W. File No. 2397, 1930, City Hospital. Referred by Dr. K. Boy aged sixteen years. Admitted to City Hospital October 30, 1930.

History of Injury: Shot his hand with a blank cartridge, causing burn in the palm of hand. Seven days later he noticed some stiffness in muscles of jaws while he was at dinner. This soreness gradually became worse and he was admitted to the hospital October 30, nine days after the accident and two days after the first discomfort was noticed in the muscles of his jaw.

Examination: On admittance to hospital we found a boy sixteen years old, well nourished. He has a powder burn of the hand and some swelling. Also he has a vaccination for

smallpox that was ten days old and causing no trouble. The boy complained of soreness of the muscles of the jaw. Temperature 100.8°F., pulse 94, respiration 22. Blood count 11,600, 78 per cent polymorphonuclears.

Course: That night his temperature was 103°F., pulse 120, respiration 28. Patient complaining of stiffness of arms and very restless.

On October 31, patient very uncomfortable, photophobia, back muscles rigid, headache, general body stiffness, exaggerated knee reflexes. Temperature 103.6°F., pulse 110, respiration 26. Spastic smile, marked opisthotonos spasms.

November 1 and 2: No improvement.

November 3: Slight improvement, but still unable to take much liquid.

November 5: Patient still very uncomfortable, complaining of pain in back and neck, but temperature, pulse and respiration very nearly normal.

November 7: Still has great pain in back and slight spasms. Carbolic acid discontinued because of very dark, cloudy urine which showed a shower of casts.

November 9: Still suffering with back and neck and some spasms. Very rigid body.

November 10: Much more comfortable. Sodium amytal, which had been given the patient daily, discontinued.

November 12: Much better; some stiffness.

November 13: Carried before the hospital staff in rolling chair.

November 14: Discharged. Still has some limitation of motion due to disfunction of affected muscles.

This patient did get 1/8 grain of morphine on his first night in hospital, but no more. His urine cleared up within a few days.

Case IV. E. B. File No. 1147, City Hospital, 1931. Aged eleven, admitted to hospital June 4, 1931.

History of Injury: On May 26 stepped on rusty wire in barn. Nothing was done that night. The next morning it was washed and iodine applied. On May 31, just five days after the injury, patient noticed he could not eat without some pain, especially when he tried to open mouth wide. June 1, he had difficulty in swallowing. Patient admitted to the hospital June 4.

Examination showed a healthy eleven year old boy, unable to open mouth or talk well. Tongue protruded between the teeth with difficulty. Examination of throat by specialist showed enlarged tonsils, but no acute infection. Chest normal. This patient had no other muscular rigidity with the exception of slight opisthotonus. All reflexes were exaggerated.

Course: This patient did not get intraspinal antitoxin, but other treatment was the same.

June 8: Patient much better, able to talk.

June 9: Patient allowed up on chair and for financial reasons was allowed to go home on June 10.

For two weeks after he left the hospital he was unable to walk, but at the end of the month there was no evidence of such spasm.

Case V. J. J. File No. 1483, City Hospital, 1931. Referred by Dr. T., aged twenty-four. negro. Admitted to hospital July 12, 1931.

History of Injury: Colored man with no history of injury, but we removed a splinter from plantar surface of foot. History given by members of family is that five days ago he had sore throat and stiffness of muscles of neck and jaws which got worse until July 11, he began to have convulsions with white frothy sputum pouring from mouth. The convulsions occurred every hour and lasted from two to four minutes; he was conscious between spasms. The family on being asked why they had not brought him sooner said they were waiting for a brother to come who was out of town.

Examination: On admittance Sunday morning, July 12, the first impression was that patient was in hopeless condition, especially after a convulsion lasting for fully five to eight minutes. Spastic smile. Skin dehydrated. Restless, but partly conscious, a board-like rigidity of all muscles. Temperature 104°F., unable to get pulse, respiration 58, shallow and labored respiration. Blood count 10,350, 88 per cent polymorphonuclears. Urine four-plus albumin, cloudy.

Course: Full treatment was given, with no improvement. Pulse was never under 160. Convulsions continued. Patient died 1:30 A.M. July 13.

I believe this man could have been saved if he had been brought to the hospital five days before when the disease first manifested itself. This is a death due to ignorance; all the negroes believed he had been "conjured."

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FIBROMYOMA OF THE RECTUM

REPORT OF A CASE*

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HUNT[1] in 1921 reviewed the literature concerning myomas of the rectum, found 20 cases and added



FIG. 1. Gross specimen illustrating character of tumor.

4 from the Mayo Clinic, 2 fibromyomas and 2 myomas. These benign tumors commonly give few signs until their increasing size causes obstructive symptoms. Their surface may become eroded and bleed and pain in the rectum is a common complaint of patients with these tumors.

These neoplasms are frequently pedunculated and present in the bowel lumen, although they may be extrinsic, and, as they grow larger, may decrease the caliber of the bowel.[2] They apparently originate in the muscularis mucosa or muscularis.

I wish to add another case report to those already in the literature.

CASE REPORT

Mrs. C. B., aged fifty-four years, presented herself for examination, complaining of long-standing constipation with increasing difficulty in moving the bowels; a sensation of a lump in her rectum; loss of 37 pounds in weight, and urinary frequency. Ever since a hysterectomy performed eight years ago, it has been necessary for her to take more and stronger laxatives. Within the last three months if her stools were not thin and watery, no bowel movement could be obtained. During this time she had a sensation of a lump or mass in her rectum. Walking or being on her feet increased this discomfort. About a year before examination, she had a severe hemorrhage from the rectum.

Examination revealed a firm pedunculated nodular tumor filling the rectum, attached along the left margin and posterior wall, from near the ampulla to within a few centimeters of the sphincter muscles.

Roentgenograms and fluoroscopic examination of the colon and rectum revealed a smooth pedunculated mass, apparently a benign tumor. A biopsy was performed through a proctoscope and microscopic examination of the tissue revealed a vascular fibromyoma. Other laboratory and physical examinations were unimportant.

Removal of the tumor was proposed. The patient entered the Wells County Hospital and was given a low residue diet. An ounce of liquid petrolatum was taken three times a day, as well as daily enemas. The patient was kept in bed and vaccination for peritonitis was performed according to the method of Bargen and Rankin.[3] After about a week of this regime, rectal examination revealed that the tumor had shrunk approximately a third in size and by inserting two fingers in the vagina, the tumor could be made to present at the anus.

With the aid of ethylene anesthesia, and with two fingers in the vagina and one in the rectum, the tumor was forced downward by gradual pressure, until it presented through the anal rings. The pedicle was clamped, cut, ligated, and the stump covered over with rectal mucosa.

The convalesence was uneventful and the patient left the hospital within ten days. Incontinence did not develop, and there was

* From the Caylor-Nickel Clinic. Presented before the Association of Residents and Ex-residents of the Mayo Clinic, Oct. 9, 1931, Rochester, Minn.

no sign of recurrence nine months after the operation. Much of the weight lost has been regained.

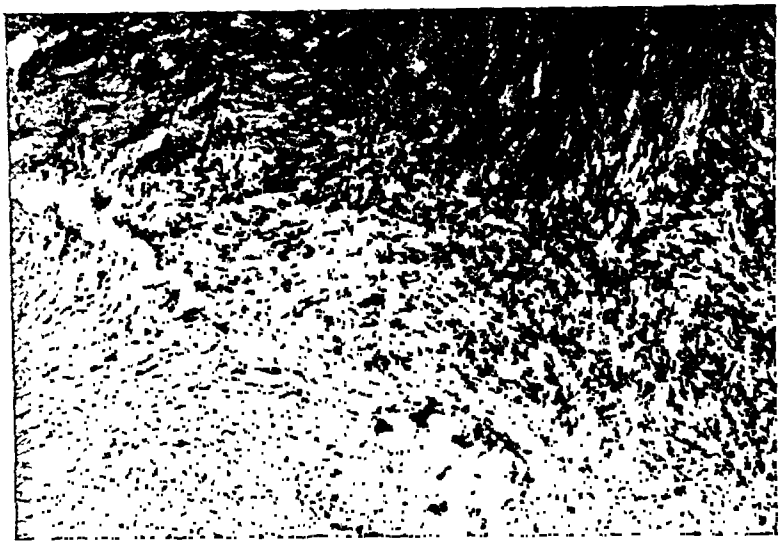


Fig. 2. Microscopic features of tumor include whorls of fibrous connective or muscle bundles, many thin-walled blood vessels and areas of degeneration. (× 100.)

The tumor at the time of its removal weighed 110 gm. and measured 7 by 6 by 4.7 cm. (Fig. 1). It was lobular and about two-thirds of its surface was covered by eroded, hemorrhagic mucosa. At one end of the tumor was a yellow to gray sloughing area, the site of the recent biopsy. Cut surfaces revealed interlacing whorls of grayish white fibrous tissue with a few yellow to gray areas of necrosis. The cut surfaces bulged after release of the intratumorous tension.

Microscopic examination revealed bundles of fibrous connective tissue and muscle as the chief structures that composed the tumor. There were areas of necrosis and many large blood spaces were seen (Fig. 2). Dr. Broders has examined slides of the tumor and concurred in the diagnosis.

Surgical removal of tumors such as this is oftentimes a difficult technical procedure. If the growth is pedunculated and can be made to present through the anus, so a visualized conduct of the operation is possible, their excision is easy. But if the lesions are high lying and attached by a short pedicle, other procedures must be considered such as fulguration, pressure forceps, ligation, clamp and cautery excision, colotomy or resection.[4]

In this case, the preoperative preparation was a most valuable aid in the successful culmination of this operation.

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RECONSTRUCTION-ARTHROPLASTY OPERATION FOR THE HIP*

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IN a twenty-year association with Dr. Royal Whitman at the Hospital for Ruptured and Crippled, I have learned from observation and a considerable personal experience, that the reconstruction operation devised by Dr. Whitman has a very extensive application in certain difficult hip lesions. The essential features of the reconstruction operation are: (1) Removal of all obstructions from the acetabulum, such as the femoral head in ununited fracture through the neck, the dense soft tissue which fills the acetabulum in resistant cases of congenital dislocation of the hip, the numerous bony masses within the joint capsule and about the acetabulum in osteoarthritis, and so forth. (2) Remodelling of the neck of the femur or of the upper extremity of the femur into a form that approximates a femoral head and neck. (3) Osteotomy of the greater trochanter and implantation of this process lower down on the shaft of the femur to permit abduction of the limb.



FIG. 1. CASE III.



FIG. 2. CASE III.

The primary result of this operation is stability of the hip and the limb. Pain is usually greatly diminished. Mobility of the joint is a variable factor. In some cases there may result as much as 30 or 40 degrees of flexion, 20 degrees of abduction and the same degree of rotation. However, in the majority of reconstructed hips there

* From the author's service at the Hospital for Joint Diseases. Submitted for publication November 25, 1931.

is little motion, and not infrequently there is none. In addition, there is a strong tendency to a flexion and adduction de-

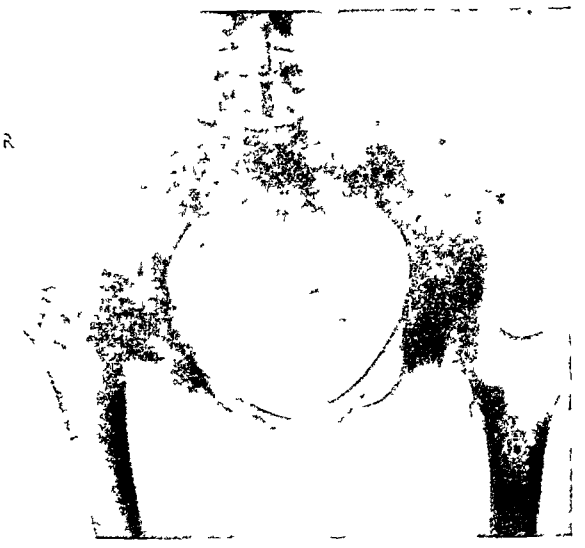


Fig. 3. Case v.

formity with an inevitable limp and consequent disability. The restricted motion adds to the disability. Discomfort in the hip is at times present to an annoying degree. When one recalls that the newly constructed head is bare of cartilage and rough, one realizes that a fibrous union is bound to occur, and, as in other joints with fibrous healing, pain, deformity and limited motion follow.

It therefore occurred to me that the reconstruction operation could be made more useful and more widely applicable by combining with it, on all occasions and at all ages, the advantages of an arthroplasty. The operation which I now perform is a typical Whitman reconstruction operation, but in addition I cover the newly formed femoral head with a double layer of fascia lata removed from the same or the opposite limb, and secured by sutures that pass through the substance of the adjacent bone.

Technique of the Reconstruction-Arthroplasty Operation

A half U incision is made over the antero-external aspect of the hip. The line of cleavage between the tensor fascia femoris and the gluteus medius muscles is found and the muscles are retracted laterally.



Fig. 4. Case v.

The capsule of the joint is now in view. With an osteotome the greater trochanter is severed from the femur. Care must be exercised to cut the trochanter at such an angle that there will be ample substance to form a head and fairly long neck. The trochanter and the pelvitrochanteric muscles are retracted. The capsule is incised and its edges are separated sufficiently to give a free exposure of all the structures of the hip. The next step in the technique depends upon the existing pathology. If the operation is for an ununited fracture of the neck, the femoral head is removed and the stump of the neck rounded off and filed to give a globular even extremity. If the condition is an osteoarthritis, the overgrown head is removed completely or nearly so, and the extremity of the neck or what is left of the head is made smooth with a chisel and file. If the operation is

for an ankylosis, an osteotomy is done in the vicinity of the joint line, the limb is everted and adducted, dislocating the upper extremity of the femur, which is remodelled into a head and neck. The acetabulum is enlarged with a reamer.



Fig. 5. Case vi.

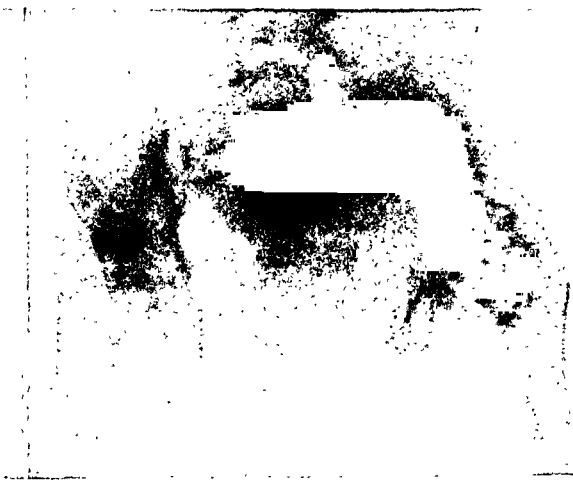


Fig. 6. Case vi.

A 5 inch vertical incision is then made on the lateral aspect of the thigh beginning at the bottom of the original U incision. This exposes the fascia lata, from which a strip about 6 inches long and 4 inches wide is removed. The latter is placed in saline solution.

Following the suggestion of Dr. Campbell, two drill holes are made through the base of the newly formed femoral neck, one on either side. The fascia is placed over the femoral head and folded back on itself so that there are two layers of tissue between the acetabulum and the newly formed head. The fascia is secured to the femoral head by chromic catgut sutures that pass through the drill holes. It is further held down by a purse-string suture which goes around the neck. The femoral head is placed in the acetabulum and held there by abducting the limb.

The periosteum and some cortical bone equal in area to the greater trochanter are removed from the outer surface of the femoral shaft just below the neck. The greater trochanter is contacted with this raw surface on the femur and held there by numerous interrupted strong catgut sutures which pass through the periosteum over the trochanter, the periosteum and bony tissue lifted from the femur, and the aponeurosis of the vastus externus. The wound is closed in layers, interrupted catgut sutures for the deep tissues, chromic catgut for the deep fascia, plain catgut for the superficial fascia and silk for the skin, being employed.

Prior to the beginning of the operation moleskin adhesive plaster stickers for traction are applied to the leg from the ankle to a little above the knee. When the operation is completed a plaster-of-Paris spica bandage is applied, holding the limb in about 25 degrees of abduction, 15 degrees of flexion and slight inward rotation. The spica extends from the nipple line down on the affected side to near the ankle.

As there is always some oozing of blood after almost any hip operation of fair magnitude, it is helpful to insert a rubber tissue drain which can easily be removed after forty-eight hours.

The patient is returned to his bed, the head of which has been elevated on stilts about 15 inches high. The surface of the bed is rendered smooth and unyielding by inserting a large board between the spring and the mattress. Traction is applied im-

mediately; the amount depends upon the size and the tolerance of the patient. Five to 10 lb. of traction at the beginning are sufficient. This can be increased to 15 to 20 pounds in a few days.

The traction and plaster are left on for five weeks. Enough time must elapse to permit healing of the trochanter to the femur. The plaster is then removed and a swinging traction posterior splint is applied to the limb from the groin to the foot. Baking and massage of the hip and thigh and light passive movements are instituted. Active motion of the hip can be started a week later. Walking should be attempted at the end of about eight weeks after the operation.

The advantages of this procedure over the ordinary reconstruction operation are: (1) Pain is reduced to a minimum because the newly formed head is not only made as smooth as possible, but it is covered with a double layer of fascia. (2) The interposition of fascia between the head and acetabulum assures a fairly extensive range of motion in the hip joint.

Stability of the hip is as secure after the reconstruction-arthroplasty operation as after the simple reconstruction operation.

CASE REPORTS

Case 1. M. O., fifty years of age, was admitted to the Hospital on February 9, 1931 and discharged April 21, 1931. This man complained of pain in both hips, but more severe in the left. The pain in the left hip appeared about three years ago and was not preceded by any known injury or illness. The pain was followed by disability in walking. The symptoms increased and became so severe that the patient had to resort to the use of crutches.

The examination showed a bilateral osteoarthritis of the hips, worse on the left side. The man walked with great difficulty and with a marked limp. The left lower limb was flexed at the hip to 160 degrees, adducted 15 degrees and was in outward rotation. Rotation and abduction were completely restricted. There was a range of flexion from 160 to 125 degrees. The *x*-ray picture showed marked enlargement of the femoral head and acetabulum, numerous bony overgrowths on the femoral head and neck, and a reduction in the joint space. The right hip showed a similar lesion. As the left hip and lower limb were more painful and caused greater inconvenience than the right, it was decided to operate upon this joint first.

Reconstruction-arthroplasty operation on February 13, 1931. The technique used was that already described. The head of the femur was found to be enormously enlarged. Its surface was irregular and had numerous areas of atrophy of the cartilage. There was much overgrowth of bone. Most of the head was removed; the remainder was converted into a smooth globular extremity. The acetabulum was smooth except for some masses of fibrous tissue near its lower pole. A fascial flap was taken from the same thigh, doubled up and securely fixed over the new femoral head.

This man had an uneventful convalescence. The plaster spica was removed in a month. Two months after the operation there was a fair range of motion in the hip. The patient was fitted with a Thomas brace and ring and discharged.

Result. October, 1931: The globular extremity of the neck now fits very well in a roomy acetabulum. Clinically this patient has greatly benefited by the operation. He has practically no discomfort in the operated hip. There is a range of motion of about 70 degrees of flexion, 25 degrees of abduction, and fairly free rotation.

Case 11. H. P., seventy years of age, was admitted to the hospital on April 23, 1931 and discharged on July 14, 1931. Her chief complaint was severe pain in the right hip and total inability to walk or even stand. On February 8, 1931 she fell and broke her hip. The abduction treatment was given but could not be carried out efficiently because of lack of coöperation of the patient. The plaster spica was removed against advice by the surgeon taking care of her.

Examination showed that the patient was thin but in good general condition. She was unable to walk. Manipulation of the injured limb was very painful. The right lower limb was evidently shortened. It was in outward rotation, adducted, and flexed at the hip to 150 degrees. Measurement showed that the right lower limb was 1¼ inches shorter than the left. The *x*-ray picture revealed a fracture of the neck of the femur without union.

Reconstruction-arthroplasty operation was performed on April 24, 1931 under ethylene

anesthesia. The wound healed by primary union. The result seemed most satisfactory from every standpoint, but I had the same experience as her first physician. She was uncooperative and most refractory, refusing to help herself. The postoperative *x*-ray picture showed a satisfactory reconstruction. Finally this patient began to walk, but because of her disagreeableness was transferred to a convalescent home.

Case III. C. B., fifty years old, was admitted to the hospital on April 27, 1931 and discharged August 13, 1931. On January 22, 1931, he was struck by an auto and sustained a fracture of the left hip. He received indifferent treatment and the fracture did not heal.

Examination showed that he was markedly disabled. He just managed to get about with two crutches, but he could not bear weight on the left leg. He was thin, pale and in poor general condition. The left lower limb was 1¼ inches shorter than the right. It was adducted and rotated outward in an attitude characteristic of fracture of the hip. The *x*-ray picture (Fig. 1) showed an ununited fracture of the femoral neck with upward displacement of the shaft. As the fracture had not united in four months, it was unlikely to heal and an operation was advised.

The reconstruction-arthroplasty operation was done on May 1, 1931. Due probably to this man's poor general condition a mild wound infection with Staphylococcus aureus ensued. The postoperative routine was however not disturbed. The limb was placed in a swinging traction apparatus six weeks after the operation. At this time he had 30 degrees of flexion and 25 degrees of abduction. At the time he left the hospital he walked fairly well with one crutch. As he is now somewhere in Pennsylvania I have been unable to find out his present condition. The postoperative *x*-ray (Fig. 2) shows a satisfactory reconstruction. There is every prospect of a good result.

Case IV. H. L., fifty-three years of age, was admitted to the hospital on May 18, 1931 and discharged August 12, 1931. His chief complaint was pain in the right hip and knee and increasing difficulty in walking. On July 24, 1929 he was thrown from a second story window and fractured his right hip. When I first saw him, nearly two years later, he walked with a limp on the right side and with the aid of two crutches. The right lower limb was in marked outward rotation and moderate adduction. There was 1½ inches' shortening. The *x*-ray picture showed an ununited fracture of the neck of the right femur with extensive absorption of the neck and upward displacement of the femoral shaft.

On May 22, 1931 he had a reconstruction-arthroplasty operation. After removal of the femoral head the stump of the neck was found to be exceedingly small, hence the newly formed head was of very small dimensions. He stood the operation well but developed cardionephritic symptoms including dyspnea, irregular weak heart action and edema of the limbs. He had also a mild wound infection but without extrusion of the fascia. The cardiac lesion and the infection prolonged his stay in the hospital.

At present he is walking about fairly well. He has no discomfort in the operated limb. He still uses crutches. This is due to pain which he has in the left, unoperated, leg. The operated limb is in slight inward rotation. He is able to bear weight on it. There is unusually free motion in the hip, including flexion from 180 to 120 degrees, 25 degrees of abduction and marked inward rotation. A recent *x*-ray picture shows the newly formed head rather small for the acetabulum, but well within the upper part of this cavity.

Case V. M. R., twenty-five years of age, consulted me in 1925 for shortening of the right lower limb, a hollow back and difficulty in walking. At the age of two years she had pneumonia which was followed by an arthritis, probably suppurative, of the right hip. Shortly thereafter the parents found that the patient limped and that her right leg was shorter than the left. The deformity of the right limb gradually increased so that each year she had to increase the lift in the right shoe. At the same time the hollow in the lower part of her back increased.

When I saw her she walked with a marked limp. The right lower limb was flexed and adducted, rotated outward at the hip and shortened. There was a marked lordosis. There was also marked atrophy of the limb. The *x*-ray showed what I took to be a pathological dislocation of the right hip probably secondary to a suppurative epiphysitis or arthritis. I advised a reconstruction operation.

The patient left me and returned six years later, in June 1931, with all the symptoms aggravated. The deformity had become marked. The gait was very awkward. The

limb was fixed in an attitude of outward rotation, adduction of at least 15 degrees and flexion to 120 degrees. An *x*-ray picture (Fig. 3) showed complete disappearance of the right femoral head, flattening of the neck, upward displacement of the femur and apparently disappearance of the acetabulum. The advice to have her hip operated upon was accepted.

On June 9, 1931, a reconstruction-arthroplasty operation was performed. Very slight motion was found to be present. Contrary to expectation the femoral head was not absorbed. It was present but much flattened and elongated with overhanging edges. There was practically no neck. Its surface was smooth and covered with a tissue that looked like thin cartilage. There were numerous areas on the head from which the cartilage had disappeared; there were others from which fibrous tissue projected. The latter accounted for the ankylosis. The acetabulum was shallow and covered with a tissue that resembled abnormally thin cartilage. The ligamentum teres lay across the acetabulum and was greatly thickened. It was removed. The femoral head was reduced to about a fourth of its size. Enough bone was removed from its sides to somewhat resemble a femoral neck. The acetabulum was enlarged with a reamer. The newly formed head was then covered with fascia and the operation completed as already outlined.

This patient had an uneventful convalescence. The deformity was corrected. The postoperative *x*-ray picture (Fig. 4) shows a satisfactory reconstruction. At present (October, 1931) she walks about without any aid. The operated limb is parallel to the opposite leg. There is about 10 degrees of abduction and 40 degrees of flexion in the operated hip. The limb is painless and is rapidly improving in its function of weight bearing.

Case vi. I. S., six and one-half years old, came under my care on August 16, 1927 for suppurative arthritis of both hips. This child was well up to the age of nine months when, following a fall, the right hip became infected. Some short time later the left hip too became infected. When I first saw this child, at the age of two and one-half years, she had a low grade suppurative arthritis of both hips with huge abscesses in the thighs and fixation of both hips. The abscesses were evacuated and the limbs immobilized. In the course of several months the inflammation and the infiltration about the hips subsided and the sensitiveness disappeared. The child resumed walking. Both hips were ankylosed with complete destruction of the hip joints.

In May, 1931, the child had been free from symptoms for about three years. She had had two stretchings for correction of deformity at the hips without any recurrence of the inflammation. The hips were ankylosed (Fig. 5). It seemed very likely that the infection had completely disappeared. It was, therefore, deemed safe and opportune to re-establish at least one good hip and an operation was advised.

A reconstruction-arthroplasty operation was performed on the right hip on June 2, 1931. The technique already described was employed. Considerable difficulty was encountered in moulding the upper extremity of the femur into a head and neck. The acetabulum was filled with bone and soft tissue and had to be reformed by a reamer. The ultimate result from an anatomic standpoint was satisfactory. The postoperative *x*-ray picture (Fig. 6) shows a very fair hip joint.

Clinically this patient has a good result inasmuch as there is a painless range of motion in all directions. There is about 25 degrees of flexion, 15 degrees of abduction and rotation. Walking is not so good as it might be because of the fixation of the unoperated hip in flexion and adduction. Nevertheless, the child does walk very well and the operation can be considered successful.

Case vii. J. S., thirty-three years old, consulted me for increasing pain in the right thigh and an increasing limp. He was apparently well until six years ago when he began to experience a tired feeling in the right thigh. Pain and a limp supervened and gradually increased.

Examination showed a vigorous man who walked with a marked limp on the right side. The right lower limb was flexed, adducted and rotated outward at the hip. Inward rotation was completely restricted. An *x*-ray picture showed an enormous enlargement of the head of the right femur. The articular surface of the head was irregular. The joint space was greatly reduced. There was a marked overgrowth of bone at the upper part of the acetabulum. This man evidently had an extensive osteoarthritis of the right hip joint for which a reconstruction-arthroplasty operation was advised.

The patient was admitted to the Hospital for Joint Diseases on April 22, 1931; he was operated upon two days later and was discharged to the care of his family physician on May 4, 1931. The typical operation was performed. No technical difficulties were encountered. The femoral head was found to be about 2½ times the natural size. The articular cartilage both on the femoral head and in the acetabulum had numerous atrophic areas. In many spots there was complete disappearance of the cartilage. The femoral head was reduced in size until it was somewhat smaller than the normal. It was then thoroughly covered by a double layer of fascia lata. The limb was immobilized in a double spica which extended to the ankle on the right side and the knee on the left side. Traction was applied as soon as the patient was returned to his bed.

Ten days after the operation the patient, for financial reasons, insisted that he be discharged and that the rest of the postoperative care be given him at home by his family doctor. During his stay in the hospital he seemed well. He had very little postoperative discomfort, slight febrile reaction for a day or two, and no unusual complaints.

On the seventeenth postoperative day, one week after he got home, he suddenly experienced precardial oppression, became cyanotic and dyspneic and in a few hours expired, presumably from a pulmonary embolism. The postoperative *x*-ray picture of the hip showed a satisfactory reconstruction of the hip.

SUMMARY

The reconstruction-arthroplasty operation is an outgrowth of the Whitman reconstruction procedure. The technique employed is that described by Dr. Royal Whitman plus the use of a double layer of fascia lata to form a sort of bursa between the newly formed femoral head and the acetabulum. The fascia is secured to the femur by sutures passing around the neck of the femur and through drill holes in the base of the neck. It has so far been used for ununited fracture of the neck of the femur and osteoarthritis of the hip for which the Whitman reconstruction operation is chiefly intended. It has, however, also been applied in two cases of ankylosis of the hip in which this operation has the advantage over an ordinary arthroplasty in that the trochanter was displaced downward thus securing a fair range of abduction. The reconstruction-arthroplasty, which combines the technique of the reconstruction operation and hip arthroplasty, is indicated where either of these procedures is necessary, and is likely to prove more effective than either because of the additional technical features.

Seven patients, varying in age from six to seventy-five years, have been operated upon. There were 3 cases of ununited fracture of the hip, 2 cases of hypertrophic osteoarthritis of the hip, and 2 cases of ankylosis of the hip, one of two years' duration and one of twenty-four years' duration. The results, while not final because of the short time since the operation, are very encouraging.

PERIPHERAL VASCULAR SYSTEM IN THE CHRONIC ARTHRITIDES*

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THIS investigation is designed to determine the clinical status of the peripheral vascular system in the chronic arthritides. The cases have been classified as follows:

1. Osteoarthritis (syn.: hypertrophic or degenerative "type")
2. Rheumatoid arthritis (syn.: arth. deformans or atrophic "type")
3. Non-specific infectious arthritis (syn.: proliferative "type").

Osteoarthritis and rheumatoid arthritis we consider to be distinct morbid entities, having a clinical course and pathology peculiar to themselves. The third category is a heterogeneous group in which at present no further differentiation is possible, although the probabilities involve several types of streptococcus. I agree with Archer[1] and Cecil that the so-called arthritis of the menopause is osteoarthritis, but in addition we must emphasize the frequency of combined arthritis in this age group.

I shall not at this time attempt a comprehensive exposition of the pathology or symptomatology of these respective conditions, but confine myself to observations on the vascular mechanism, intending thereby further to differentiate the two distinct diseases from the heterogeneous group.

Two hundred cases of chronic arthritis were recently examined. Another hundred patients in whom the only or chief complaint was varicose veins were questioned for rheumatoid histories and examined for signs of joint pathology. Thirty-two patients with obliterative vascular disease (thromboangiitis obliterans and dysbasia angiosclerotica) were similarly examined and questioned. To supplement the clinical observations specimens of tissues obtained at the operating table from arthritic joints, and sections of limbs amputated because of obliterative vascular disease were also studied for the histopathologic appearance of the articular and periarticular vascular bed.

OBSERVATIONS

1. *Blood Pressure.* Most monographs which discuss the blood pressure readings in the chronic arthritides state that it is low or a low normal.[2] In our series this is true for only one category, rheumatoid arthritis. The contrary is true for osteoarthritis. In non-specific infectious arthritis the systolic pressures, while higher than the accepted average normals, are well within the limits of normal variation. The figures of Symonds, quoted by Fishberg,[3] were used as average normals. According to the latter, a systolic pressure above 150 mm. "is to be regarded as abnormal at all ages." In Table 1 it will be noted that in each age group over forty years the average systolic pressure in osteoarthritis is definitely hypertensive. The following figures will present a more accurate picture. Over 90 per cent of our osteoarthritics had a systolic pressure well above the average normal for their age group, and 61 per cent were above the 150 mm. limit. The apparent aberration in the forty to forty-four year age group of infectious arthritis is caused by the preponderance of females in the menopause age. The frequency of combined arthritis

* From the Service of Dr. Samuel Kleinberg, Hospital for Joint Diseases, and the Orthopedic Services of Dr. P. W. Nathan, Montefiore Hospital, and Mt. Sinai O. P. D. Read before the Section on Orthopedic Surgery, New York Academy of Medicine, November 20, 1931.

at this age is also a factor. The remarkably low pressure in rheumatoid arthritis coincides with the observations of others. In the appended graph (Fig. 1) these relationships will appear more obvious.

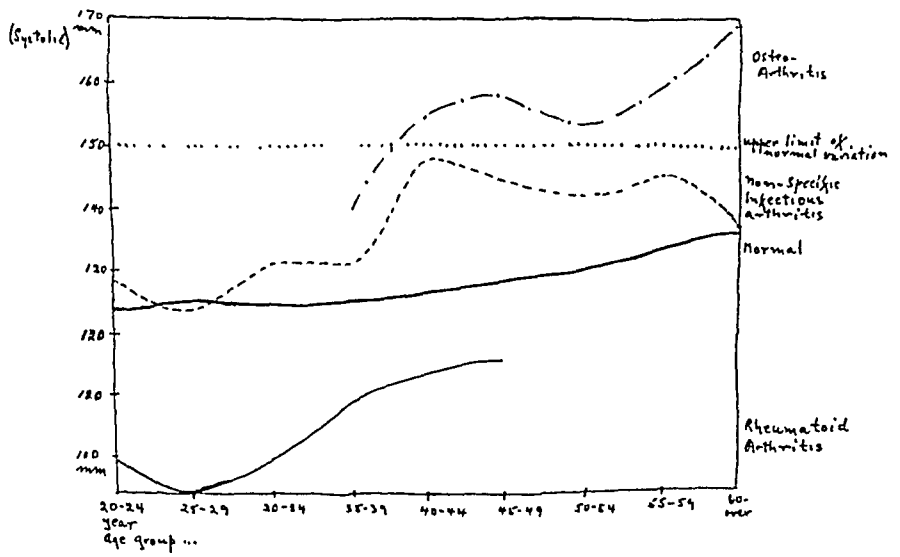


FIG. 1. Respective curves of averages for systolic blood pressure in three arthritides compared to accepted normals; 150 mm. marks upper limit of normal systolic variation; above this figure systolic pressure is considered abnormally hypertensive.

The tendency toward hypertension in osteoarthritis is intimately related to its symptomatology. Quoting Norris:[11]

> Muscular cramps especially in the lower extremities and at night is a common and annoying symptom of hypertensive people. The individual is often awakened by an intensive cramp in the calves excruciating in severity and relieved by active function, heat, or, if he can accomplish it, walking . . . The attacks are somewhat like those of intermittent claudication but the latter are induced by exercise and relieved by rest. The former occur during rest and are relieved by exercise . . .

With very little modification this picture will be familiar to anyone who has taken histories of moderately advanced cases of osteoarthritis. It is a frequent experience that in patients in whom osteoarthritis is accompanied by excessive blood pressure, relief from joint pain can follow sedative (as distinct from analgesic) medication.

2. *Obliterative Vascular Disease.* Thirty-two cases of obliterative vascular disease were examined in the Clinic for Vascular Diseases of Dr. Samuel Silbert at Mt. Sinai Hospital. No attempt was made in this study to differentiate between thromboangiitis obliterans and dysbasia angiosclerotica for two reasons: (1) differentiation in the middle age groups is difficult, and (2) the effect on the articular tissues is in both instances the same, that of a diminution of arterial blood supply. The average age of these patients was 42.2 years, a great majority ranging from thirty-eight to fifty. Of these only one presented a sign of osteoarthritis and this was an asymptomatic Heberden node. Four cases offered histories of previous transient rheumatic pains in joints other than those involved by the present complaint.

To correlate our arthritic patients with the foregoing observation the dorsal pedis pulsation was recorded in most of the examinations. In osteoarthritis the pulse was invariably of an intensity within normal range. In rheumatoid arthritis, however, a tendency toward diminution was noted progressing in most instances with the advance of the disease process. In its later stages, an absence of the dorsalis pedis pulsation was not infrequently ob-

served. On the Orthopedic Service in Montefiore Hospital there are always several cases of end-stage rheumatoid

Table 1

Age		Systolic	Diastolic	Pulse Press.
20–40	Normal	124.2	80.5	43.7
	Osteo-arth.			
	Rheumatoid arth.	100.0	70.0	30.0
	Infec. arth.	128	63.0	65.0
25–29	Normal	125.5	81.5	43.0
	Osteo-arth.			
	Rheumatoid arth.	95.0	70.0	25.0
	Infec. arth.	124.0	78.0	46.0
30–34	Normal	125.1	82.3	42.8
	Osteo-arth.			
	Rheumatoid arth.	100	68	33
	Infec. arth.	132	79	53
35–39	Normal	125.3	83.3	42.0
	Osteo-arth.	140.5	88.0	52.5
	Rheumatoid arth.	110	60	50
	Infec. arth.	130.8	74.5	56.3
40–44	Normal	126.4	84.0	42.4
	Osteo-arth.	155.0	81.8	73.2
	Rheumatoid arth.			
	Infec. arth.	148.0	77.0	71.0
45–49	Normal	128.2	84.7	43.4
	Osteo-arth.	156.8	88.3	68.5
	Rheumatoid arth.	115	80	35
	Infec. arth.	144.4	83.1	61.3
50–54	Normal	130.2	85.7	44.3
	Osteo-arth.	152.4	89.6	62.8
	Rheumatoid arth.			
	Infec. arth.	141.2	76.2	65.0
55–59	Normal	133.5	86.8	46.7
	Osteo-arth.	158.0	86.6	71.4
	Rheumatoid arth.			
	Infec. arth.	145.0	85.0	60.0
60–over	Normal	135.2	86.9	48.3
	Osteo-arth.	169.3	91.9	77.4
	Rheumatoid arth.			
	Infec. arth.	137.5	76.2	61.3

arthritis in which vascular obliteration is so far advanced that one or even several extremities have become gangrenous. When this occurs the tissue necrosis is general in the distal portion of the limb. It is not a discrete closure of the vessels to the hallux, but the entire foot or hand shows evidence of the change.

The earliest manifestation of this process is atrophy and discoloration of the skin, a result of insufficient circulation to the superficial tissues. In most instances this atrophy is self-limited and remains at a constant level for many years. However, later in life a further decrease in the patency of the vessels from any added cause, such as senile arteriosclerosis, crosses the margin of vascular sufficiency and gangrene ensues. There is undoubtedly in this composite picture of vascular obliteration in rheumatoid arthritis a neurotrophic factor, but the diffuse musculofascial fibrosis which is the effective morbid lesion plays the overt part.

3. *Varicose Veins.* In each case of arthritis the presence or absence of varicosities was noted. Seventy-seven per cent of the osteoarthritics presented varicose veins in the lower extremities. In only 28 per cent of the infectious group could they be found. These figures were compared with 100 cases from the Varicose Vein Clinic of Dr. I. S. Tunick at the Hospital for Joint Diseases. Fifty-three of these were well within the osteoarthritic age group, that is, forty years or over. Of these, about 50 per cent showed signs of the disease, 18 presenting Heberden nodes. The body habitus in patients over forty suffering from varicosities or osteoarthritis is very frequently similar, that is obese, sthenic, and characterized generally by a flabby musculature. In other words, the constitutional factor which predisposes the body to osteoarthritis likewise predisposes it to varicose veins. That the frequent presence of venous stasis occasioned by the varicosities bears a relationship to the pathology of osteoarthritis will be shown later.

4. *Histopathology.* Osteoarthritis. Sections of bone show no changes in the blood vessels. The trabeculae and marrow look well nourished. Only when erosion of the cartilage has exposed bone does any change appear in its structure, and then, in the area adjacent to the gap in the articular plate there is formed rather well demarcated granulation tissue. In the soft tissues, however, there is considerable change. The hypertrophied synovial and subsynovial tissue is decidedly hypervascular, with numerous dilated blood vessels. Perivascular accumulations of leucocytes are

frequently visible especially in the intra-articular portion where internal trauma has caused an appearance of aseptic inflammatory change. The general picture is that of vascular congestion or hyperemia of the hypertrophied tissues.[4] In those fields in which few vessels are found the fatty degeneration so characteristic of the excessively hypertrophic synovia is most evident. The easy formation of osteophytes on the articular surfaces at sites of static internal trauma is a consequence of the local hyperemia. Pathologic bone growth, whether callus or osteophyte, depends upon tissue injury and local blood supply,[5,6,7] and both conditions are favorably present in these joints.

Rheumatoid Arthritis. Sections taken at Wilson capsuloplasties were examined. These were cases in which the febrile stage had long since subsided, so that very little manifestation of any active infectious process which may have existed was present. The specimens were characterized mainly by soft tissue fibrosis in which the vessels were included, and by bone atrophy. No difference is perceptible between sections of articular bone in rheumatoid arthritis and those in obliterative vascular disease, except in degree of atrophy. The picture in the soft tissues was one of hypovascularity, with actual decrease in the size and number of the smaller vessels.

DISCUSSION

A. E. Garrod in 1907[8] with many others before and since recognized the fact that the term chronic arthritis connotes a group of diseases having in common only their localization of symptoms in or near the articulations. This is far different than the usual conception which classifies them merely as "types" of a single morbid process. The distinctions are pathologic as well as clinical. In the above sections a series of observations are recorded noting the status of the peripheral vascular system in each of these conditions. In osteoarthritis the blood supply to the extremities is characterized, generally speaking, by a sluggish venous circulation. In rheumatoid arthritis there is present a diminished arterial circulation.

The pathology of these respective diseases is what one could consistently expect from the observed vascular situation. Pearse and Morton,[7,9] Jaffe,[6,10] Muller,[5] in recent years, and others before them have shown that the physiology of bone is remarkably sensitive to blood supply. The density of the bone ends and the easy formation of exostoses near the articular surfaces at sites of internal static trauma is deducible from the presence of venous congestion in osteoarthritis.

In rheumatoid arthritis, with its gradual decrease in vascular patency, one would anticipate the marked atrophy of bone and absence of osteophytes which is actually found. We must emphasize here that the knobby, deformed hand which is seen in this disease is caused by periarticular swelling, and the subluxation of heads of metacarpals and phalanges consequent to soft tissue contraction rather than exostosis.

It cannot be said that the vascular system is an etiological factor in either of these conditions, but we wish to demonstrate that the progress of deformity, the symptoms and the disability are directly influenced by the state of the circulation and this is an integral part of the disease processes. The practical application of these facts to therapy must be obvious. The necessity for the prolonged use of measures intended to adjust the blood supply to the extremities is an important part of any therapeutic regime. In the chronic arthritides, as in many other illnesses of insidious development, it is the usual fact that the patient is not seen by the doctor until the process has become well advanced. Eradicating an etiological factor is by that time only a part of the necessary treatment. Rehabilitation of crippled limbs is equally urgent. It is here that the vascular status requires our careful consideration.

SUMMARY

1. The systolic blood pressure in osteoarthritis is, in most cases, considerably above the normal for each age group. In rheumatoid arthritis it is definitely subnormal. In non-specific infectious arthritis there is no deviation from normal variations.

2. Gradual occlusion of the peripheral arteries is a characteristic observation in rheumatoid arthritis. In advanced cases this may lead to gangrene of the distal part of the extremities.

3. Osteoarthritis is a very uncommon finding in obliterative vascular disease.

4. Varicosities occur in a very large proportion of patients suffering from osteoarthritis. The latter, conversely, is frequently found in patients in whom the chief or only complaint is varicose veins.

5. The histopathologic appearance of the articular and periarticular tissues is consistent with these clinical observations and is logically deducible.

6. Rheumatoid and osteoarthritis are further differentiated by their respective vascular changes into two distinct diseases, leaving non-specific infectious arthritis as a third heterogeneous group.

I wish to record my appreciation to Dr. Samuel Silbert and Dr. I. S. Tunick for permission to use the material available in their respective clinics, and to Dr. H. L. Jaffe, Director of Laboratory at the Hospital for Joint Diseases for his kind assistance with the pathological studies.

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THE ROLE OF SYMPATHETIC NERVE SURGERY

IN VASCULAR DISORDERS OF THE EXTREMITIES*

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INTRODUCTION

SURGERY of the sympathetic nervous system is largely a product of the past ten years. While it is true that as early as 1896 cervical sympathetic ganglionectomy was performed on a large scale for various ill-defined disorders such as epilepsy, tic douloureux, migraine, glaucoma and exophthalmic goiter, yet the procedure was soon abandoned by its most ardent early supporters. Probably the chief reason for this decline in interest was dependent upon the fact that the procedure was applied empirically without sufficient experimental data to guide its usage. The reports of Royle and Hunter[1] appearing in 1924, upon the relief of spastic paralysis by ramisection, stimulated neurological investigators generally to pursue by laboratory and clinical experimentation some of the more fundamental concepts of sympathetic nerve surgery. From these researches arose the principles for the successful treatment of certain diseases heretofore considered to be incurable.

These new therapeutic possibilities were received enthusiastically by the profession. Like almost all other new advances in treatment, we are finding that our original enthusiasm was over-abundant. Some of our early expectations have not been fulfilled, while others have been exceeded by the light of further evidence. Even at the present time, we are unable to evaluate correctly the effects of sympathectomy upon certain diseases, but in general, experience has quite thoroughly established the operative indications and the probable results.

We shall consider in this paper only the effects of sympathetic nerve surgery in the treatment of vascular disorders (arterial) of the extremities.

Before proceeding with the clinical consideration of this topic, let us for a moment review the anatomical relationships between the sympathetic nervous system and the arteries of the extremities. The caliber of the arteries is in general dependent upon the state of contracture of their muscular walls. The tone of this muscular coat is supposed to be dependent upon nerve impulses passing over two sets of fibers: (1) the vasoconstrictor; (2) the vasodilator. The vasoconstrictor impulses to the upper extremities are carried through the cervicodorsal sympathetic chain, and to the lower extremities through the lumbar chain. These impulses travel by way of the white rami communicantes as preganglionic fibers to the regional sympathetic ganglia. The impulses are then carried by postganglionic gray fibers to the spinal nerves to be distributed with them in the somatic, muscular and cutaneous areas. In the upper extremities, there is a direct sympathetic supply to the large vessels, which does not pass through the spinal nerves. Perhaps, also, it is well to mention the secondary sympathetic system along the vessels as proposed by Braeucker.[2] Thus, to destroy effectively the vasoconstrictor mechanism to the arteries of the part, it is necessary to section the regional gray rami or the preganglionic or postganglionic fibers before they enter the spinal nerves for their final distribution. Section of the rami, if completely done, would of itself destroy central vasoconstrictor connections, but the danger of missing some of them led Adson and Brown[3] to introduce ganglionectomy with trunk resection as the operation of choice

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when attempting to abolish regional sympathetic action.

The presence of a neurological vasodilator mechanism is still imperfectly understood. Whether the dilatation of the arteries, after removal of the vasoconstrictor tone, is an active or passive phenomenon is still a mooted question.

GENERAL CONSIDERATION OF VASCULAR DISORDERS

It is desirable from the standpoint of treatment to classify loosely arterial disorders involving the extremities into two main groups: (1) Those of the vasomotor, or functional nature, of which there are two types: vasoconstrictor disturbances (Raynaud's disease, arthritis deformans, scleroderma, etc.) and vasodilator disturbances (erythromelalgia); (2) those of organic disease of the arteries (Berger's disease, arteriosclerosis, thrombosis and embolism). Actually, this classification is inexact, in that the first group may be associated with organic changes in the vessels themselves, and the second group may contain varying degrees of vasomotor overactivity. As a working basis, however, the classification is feasible.

So far as we know, the only beneficial effects of depriving the extremities of their vasomotor mechanism in the treatment of vascular disorders is the increase of blood supply with its resulting increase of temperature and nourishment, due to the dilatation of the arteries, and the abolition of the sweating mechanism which prevents heat loss by evaporation. Obviously, if symptoms of the disorder are primarily due to ischemia from vasomotor overactivity (spasm), abolition of the vasomotor mechanism to the part should be highly beneficial in that more blood will be carried to the part. On the other hand, if symptoms result from organic disease in the walls of the vessels themselves with little or no vasomotor overactivity in the remaining vessels, no beneficial results would be expected from sympathectomy.

The prime considerations, therefore, in the proper selection of patients for sympathetic nerve surgery are: first, to determine the degree of ischemia; second, to differentiate between organic and spastic vascular disturbances; and third, to determine the probable clinical effect of removal of the regional sympathetic nerve supply.

METHODS FOR DETERMINING THE DEGREE OF ISCHEMIA

Ordinarily, by palpation alone, one can determine the degree of ischemia fairly accurately. When the involved extremity is colder to the touch than a normal extremity under the same environment, and when it is clammy and moist, ischemia of considerable degree may be assumed to be present. For a more accurate determination of the degree of diminished blood supply, calorimetric or surface temperature studies should be made. The Stewart calorimeter is a valuable instrument in determining the local heat production and radiation of an extremity. It is time-consuming to use, however. For practical purposes, the skin temperature studies are quite sufficient to determine the degree of ischemia. It has been shown that the surface temperatures compare fairly closely with the deep temperatures, under similar environmental conditions. There are several convenient, accurate, but expensive, thermocouples on the market. For clinical work, we have found the skin temperature thermometer to be perfectly satisfactory. It is accurate, simple and reliable. Temperature readings should be made at several points on the involved parts and comparison made between readings on the uninvolved parts. (These temperature readings should be checked several times under as nearly constant environmental conditions as possible.)

DIFFERENTIATION OF ORGANIC AND SPASTIC VASCULAR CONDITIONS

The methods so far in use for the determination or organic vascular disturbances are based upon indirect evidence obtained by the temporary abolition of the vasomotor control to the parts. After vaso-

spasticity has been eliminated by one of various methods, the resultant ischemia is assumed to be due to lesions within the arteries themselves.

Several satisfactory methods are available for the temporary abolition of vasomotor control. For the past two years, we have used acetyl choline hydrobromide, given intramuscularly in doses of from 100 to 200 mg. This drug is a powerful vasodilator, and may be administered repeatedly without detrimental effects upon the patient. We have found that the postoperative skin temperatures correspond very accurately with the skin temperatures taken during the preoperative test with acetyl choline. The technique of performing the test is essentially the same as used in all other methods. Surface temperatures are recorded prior to the administration of the drug, care being taken to keep the environmental temperature constant. After administration of the drug intramuscularly, temperature readings are made at fifteen minute intervals over a period of one hour. Mouth temperature readings are made, although this is probably unnecessary, inasmuch as we have never found a general febrile reaction to the drug. The number of degrees of rise in the surface temperature may be taken as the approximate vasomotor index. It should be pointed out that in all vasospastic conditions of the extremities, particularly Raynaud's disease, scleroderma, and arthritis deformans, periods of exacerbation of the ischemia vary from hour to hour, particularly after stimulation with change of environmental temperature and emotional excitement. To obtain a true vasomotor index, it is advisable to record temperatures during the periods of angiospasm, so that the most severe ischemia may be recorded. In conditions where angiospasm predominates, the rise of surface temperature may be as high as 12° to 14°C. after the administration of acetyl choline.

It is impossible with our present knowledge to say how high the vasomotor index should be before sympathetic nerve surgery is indicated. As a working basis, we have set the lower limit as 5°C. rise in surface temperature after the administration of acetyl choline.

Another method for determining the vasomotor index is the "fever test" devised by Adson and Brown.[3] We used this test originally and abandoned it chiefly because of the discomforting reactions, often alarming, which were produced in the patients.

Another satisfactory method for determining the degree of vasospasm is by the administration of a spinal anesthetic. We have had no experience with this method as a diagnostic procedure, feeling that it is entirely too drastic to warrant general use.

Another excellent method is the injection of the peripheral nerves supplying the area under observation with novocaine, as advocated by Lewis.[4] We have used this method in connection with the acetyl choline test and have found the degree of temperature rise with both methods to be essentially the same.

DETERMINATION OF THE PROBABLE CLINICAL EFFECTS OF SYMPATHECTOMY

The determination of the vasomotor index is perhaps the most accurate method for determining the amount of relief which may be expected from sympathetic nerve surgery.

A very reliable adjunct is often obtained from the statements of the patients themselves following temporary abolition of vasomotor control by one of the methods just mentioned. If the patient is symptomatically improved following repeated doses of acetyl choline, provided, of course, the vasomotor index indicates satisfactory response, we feel much safer in recommending sympathectomy than had this symptomatic improvement failed to appear. This is particularly true in cases of arthritis and Berger's disease. If vasodilatation has been known to occur, as indicated by surface temperature studies, and the patient has had no indication of symptomatic relief, then one must be very conservative in recommending surgery.

It might be pointed out at this time that the more conservative methods for producing vasodilatation have a distinct place in therapeutics of these borderline vasospastic disturbances. The "fever test" has been used very successfully in the treatment of Berger's disease, arthritis deformans and various other peripheral vascular disorders by the Mayo Clinic group. We have used acetyl choline in the same type of cases with very satisfactory results. Even cases of impending gangrene, of the arteriosclerotic and diabetic types, have often been symptomatically improved, and in one case of arteriosclerosis, the gangrenous process was completely arrested by the use of this drug.

RAYNAUD'S DISEASE

From the day of Raynaud's original report in 1862 until the present time, this disease has been considered to be due to an overactivity of the vasomotor mechanism without demonstrable changes in the vessels. Naturally, with the advent of sympathetic nerve surgery, this condition should be one of the first to be attacked. Adson[5] was the first to report the successful treatment of Raynaud's disease by cervicodorsal and lumbar sympathectomy. The relief of symptoms was complete and had been permanent to the time of his last report. Similar beneficial results have been reported by other observers.[6]

Thomas Lewis[4] was the first to question the theory of vasomotor origin for this disease. He considered the symptoms to be due to spasm of the vessels from causes other than vasomotor overactivity; that is a lesion within the wall of the vessel itself. His researches cast considerable doubt upon the curative value of sympathectomy in the treatment of this disease.

We have treated five patients with typical Raynaud's disease during the past two years. The relief of symptoms in the lower extremities has been complete in all patients thus far treated. In only one of the five patients, however, have symptoms been completely relieved in the hands. One patient has lost all the fingers of the right hand, three of which became gangrenous following the cervicodorsal sympathectomy. In the other three patients with recurrence of symptoms, the attacks have been precipitated by a sudden change to cold weather, or by handling ice or cold objects. The severity of the attacks, however, has been lessened in each instance, and the soft tissue changes in the fingers have entirely disappeared.

Some may argue that the operative procedure in these cases was not completely done. In each instance, the entire trunk from above the inferior cervical ganglion to below the second dorsal ganglion was removed. In the patient who had recurrence of gangrenous fingers, the entire chain from the superior cervical to the third dorsal was removed in two stages. Also, this patient was subjected to multiple periarterial sympathectomies. In spite of the radical abolishment of all known pathways for vasomotor activity to the arm, the patient continued to have severe symptoms of Raynaud's disease with an ultimate loss of digits. It seems rather paradoxical that the disease should be cured in the feet and not cured in the hands. Raynaud's disease, however, is primarily a disease of the upper extremities, although the lower extremities may be and are usually involved to some extent. This difference in the severity may be responsible for the discrepancy. On the other hand, the sympathetic nerve supply to the upper extremities is much more complex than to the lower extremities. It is quite possible that due to gaps in our present knowledge, we are failing to desympathectomize the upper extremities but have succeeded in the lower extremities. Perhaps again the disease is not primarily vasomotor. If such were the case, our failure would be readily explained were it not for the fact that the feet apparently have been cured entirely in all reported cases.

We do not wish to imply that sympathectomy is not indicated in the treatment of this condition. It offers, perhaps, the best

method at our disposal. While the disease is not "cured" in the hands, and in some advanced cases not even arrested, the improvement in symptoms more than justifies the procedure.

THROMBOANGIITIS OBLITERANS

This malady correctly belongs in Group 2 in our classification of vascular disorders, because the underlying pathological process is an organic disease of the arterial walls, affecting chiefly the end arteries. If these obliterative processes were entirely responsible for the ischemic symptoms in the parts, sympathetic nerve surgery would be clearly contraindicated. As a matter of actual observation, however, the ischemic manifestations are due partly to other factors than the obliterative processes. Some degree of vasospasm of the collateral circulation is always present. The amount of angiospasm can be measured only by determining the vasomotor index as already described. If one can demonstrate, after the vasomotor mechanism has been temporarily abolished, that the part becomes warm, that the surface temperature is increased, and the patient is symptomatically relieved following the test, then permanent removal of the vasomotor tone may be considered to be indicated. It must be kept in mind, however, that the relief afforded in this type of case is symptomatic and probably will not greatly affect the course of the disease within the arterial walls. Ischemic pain is the predominating symptom of the malady. If this factor can be controlled by one of our various methods for attacking the sympathetic nerve supply to the vessels, the result will justify the procedure.

In our experience, properly selected cases of Berger's disease treated by sympathetic ganglionectomy have given very gratifying results. Relief of the incapacitating pain has been striking. Even though amputation later becomes necessary, it can be done at a lower level than would have been possible without a previous sympathectomy. In extremities where there has not been impending gangrene or indolent ulcers, the total relief of symptoms has been maintained for many months. Furthermore, we have seen cases with cellulitis, ulcerations, and indolent ulcers completely heal following sympathectomy.

ISCHEMIC CAUSALGIA

The same line of reasoning as applies to Berger's disease applies in cases of this type. If the main vessel to a part is obliterated and the collateral circulation is insufficient, removal of the vasomotor tone should result in more adequate blood supply to the part; hence, relief of symptoms. We have had one very interesting case to illustrate this situation. The man was shot through the second portion of the axillary artery, and he suffered excruciating pain in the arm, uncontrollable by any means at our disposal. Abolition of the vasomotor tone by acetyl choline gave not only a satisfactory vasomotor index but marked relief of symptoms. Removal of the regional sympathetic nerve supply resulted in a total permanent relief of ischemic pain. We have had no experience in treating causalgia secondary to nerve involvement. Theoretically, at least, such cases would not respond satisfactorily to sympathectomy.

SCLERODERMA

This unusual skin disease is characterized by marked thickening and pigmentation of the skin with atrophy of the soft tissues about the lesions. It usually involves the hands and feet but may involve other parts of the body. The involved parts are usually cold, clammy and show definite evidence of ischemia. This disease often accompanies vasospastic disturbances like Raynaud's disease, thromboangiitis obliterans, chronic deforming arthritis, causalgia, etc. In cases of this type sympathectomy has given relief of symptoms with disappearance of the skin lesions. The completeness of relief, of course, is dependent upon the underlying cause of the deficient

blood supply. The same careful studies of the vascular supply to the part are required as in the other vasospastic disorders.

CHRONIC DEFORMING ARTHRITIS

There is a certain group of young patients suffering from arthritis, who present evidence of marked ischemia as manifested by cold, clammy, sweaty, and mildly cyanosed extremities. These symptoms usually occur in the so-called arthritis deformans which is chiefly periarterial in type. The joints are usually painful, swollen and tender. There is marked atrophy of the muscles with an early tendency toward contractures. Pain is the predominating symptom. Rowntree and Adson[7] applied the principles of sympathetic nerve surgery in the treatment of this type of case with brilliant results. The application of external heat and the institution of various physiotherapeutic measures to increase the blood supply to the involved parts have been the "ages old" methods of treatment. After all, sympathectomy is merely an elaboration of the older methods by providing a constant increase of temperature and a more adequate blood supply to the parts involved. Assuming that the arthritic patient presents symptoms of ischemia and a favorable vasomotor index, the following additional factors must be thoroughly considered in order to secure the best results:

1. There should be no mechanical hindrance to joint motion such as bony proliferation with spur formation, or ankylosis.
2. Articular surfaces should be clear.
3. Peripheral vessels should be comparatively free from arteriosclerosis.
4. The febrile reaction must have subsided.
5. All other forms of medical treatment should have failed to arrest the disease.

In dealing with arthritis, the same care for the selection of the operative case must be followed as in all other vascular disorders. Perhaps, it is even more important to depend upon a statement of the patient regarding symptomatic relief afforded by repeated injections of acetyl choline. It is very difficult at times to determine whether the pain is due to ischemia or to contractures of tendons, ligaments, and joint capsules. Unless the pain is of ischemic origin, sympathectomy will be of little avail. On the other hand, if the pain is purely of ischemic origin, prompt permanent relief may be experienced. The results in the treatment of arthritis are of the best in the field of sympathetic nerve surgery according to our experience. One may expect good results only, however, if their cases are selected with diligent care.

Before closing, there are two other conditions that at times respond well to sympathectomy and deserve mention. Slowly healing stumps with ulceration are often benefited by vasodilatation following sympathectomy. Acetyl choline in repeated large doses may occasionally serve without an operation. Cases with paralytic sequelae of anterior poliomyelitis may be materially helped by sympathectomy. If the epiphyses of the long bones have not closed, the increase of blood supply following sympathectomy will bring about a better development of the afflicted members.

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IMPROVED METHOD OF OPERATION FOR UTERINE MALPOSITION*

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THERE are 127 different operations for malposition of the uterus of which none is perfect and some are limited in use; with this in mind we have devised a method of operation that has anatomically and clinically fulfilled our desires toward the ideal.

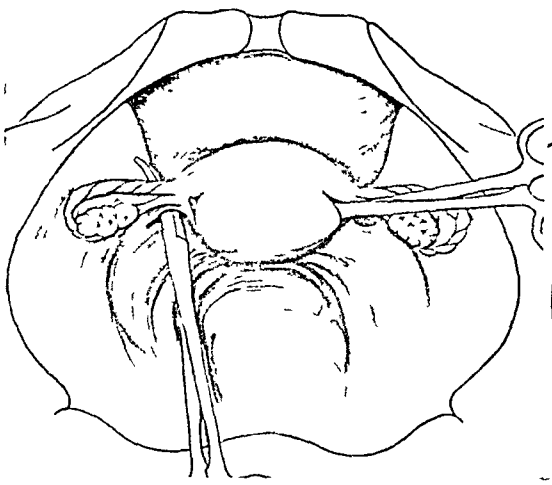


FIG. 1. An opening is made through broad ligaments. Incisions are made through serous layer of uterus. Canal is made between incisions with a mosquito or Kelly forceps.

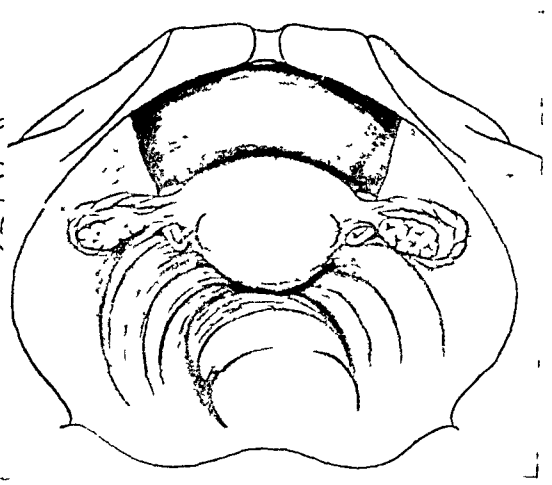


FIG. 2. Round ligaments are pulled through broad ligaments. Catgut is placed through loop of each round ligament and canal to opposite side.

Such operations as the Olshausen, Alexander, Baldy-Webster, Coffey, Gilliam, Wiley, Kelly, Mayo, Crossen, Barrett, Simpson and Montgomery are some of the many devised for this condition. Some of the objections to these operations are:

1. Recurrence of primary condition.
2. Intestinal obstruction.
3. Postoperative adhesions.
4. Difficulty of correct performance.
5. Postoperative hernia.
6. Silk stitch sinus.

We wish to change the term suspension to that of reconstruction and correction. When one studies the anatomy of the uterus and the causes of its displacements and then does one of the classical suspension operations he does do a suspension or partial suspension but the term defeats the true purpose of such operation, which in reality is a reconstruction and correction. This operation is a reconstruction of anatomical support with replacement of pelvic viscera in their normal positions.

The round, broad and uterosacral ligaments and the pelvic floor support maintain the uterus in its proper position and in relation to other pelvic organs. A second degree tear including the pelvic fascia and muscles weakens the pelvic floor retarding the normal involution of the uterus by disturbing the round ligaments or uterosacral ligaments or both, allowing a vertical or postvertical axis of the uterus with intra-abdominal pressure on the anterior surface of the uterus favors displacement which is usually posterior.

* Submitted for publication October 27, 1931.

INDICATIONS

The method of operation which I have devised is indicated in any form of uterine displacement and prolapse except in an acute inflammatory condition.

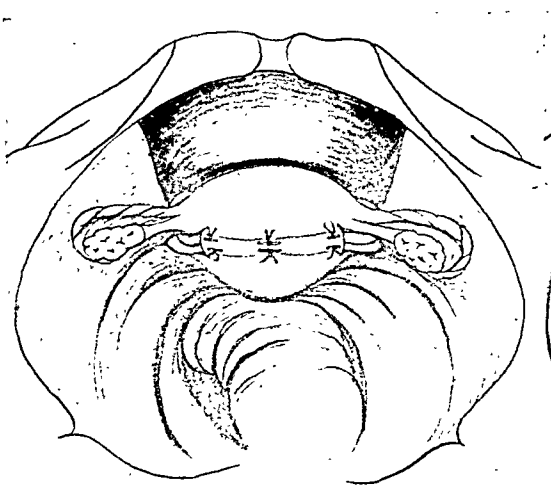


Fig. 3. Round ligaments pulled to midline and sutured.

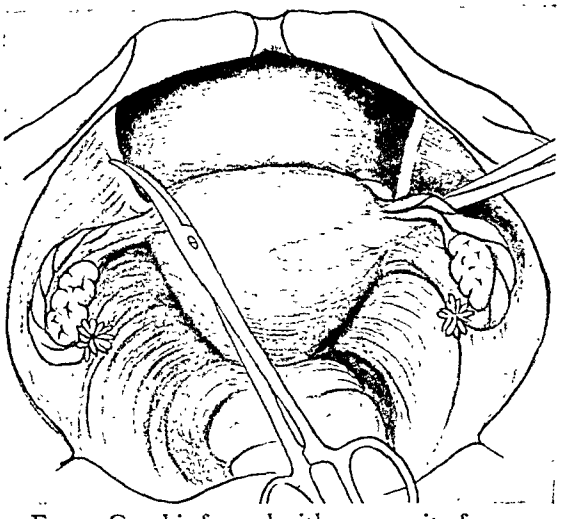


Fig. 4. Canal is formed with a mosquito forceps pushed through anterior layer of broad ligament, along posterior wall of uterus between serous and muscular layers.

TECHNIQUE

First Step. The cause is removed. This may include a dilatation and curettement, a cauterization of the cervix, repair of the pelvic floor, or the removal of part or all of one or more of the pelvic organs.

Second Step. The fundus of the uterus is lifted into position. A curved Kelly forceps is pushed from behind forward through the broad ligament close to the uterus under the tube and under the round and ovarian ligaments. A loop of the round ligament is brought back through the opening in the broad ligament. This is repeated on the opposite side. Vertical incisions are made through the peritoneum just medial to the openings in the broad ligaments or just below the level of attachment of the ovarian ligaments. A curved Kelly or mosquito forceps is pushed from one opening to the other between the serous (peritoneum) and muscular layers

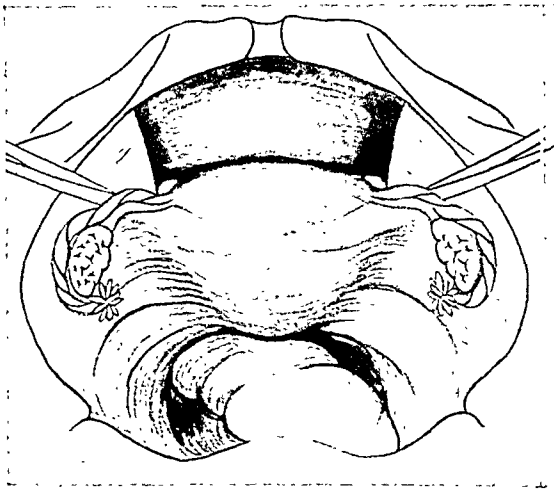


Fig. 5. Round ligaments pushed into canal to midline.

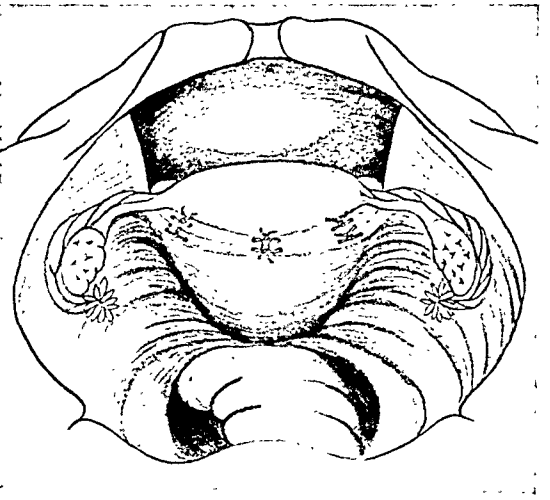


Fig. 6. Sutures in place.

of the uterus, thus forming a canal. Chromic catgut No. 2 is sutured through the loop of each round ligament and brought through the canal to the opposite side. Each round ligament is scarified. Traction is made on the catguts, pulling the round ligaments together in the center of the canal where they are sutured together with chromic catgut No. 2. The traction sutures are removed. Another suture is taken through each ligament on both sides just medial to the broad ligaments. The openings into the peritoneum are closed. (See Figs. 1, 2 and 3.)

When there is a large pelvis a second type of operation may be performed. Of both types the operation already described is the one of choice and the one we preform more often.

Operation No. 2. A curved Kelly forceps is pushed through the anterior layer of the broad ligament, then medial in the posterior wall of the uterus between the serous and muscular layer to the midline. This is repeated on the opposite side, thus forming a canal. The round ligaments are pushed into the canal with mosquito forceps. The ligaments are sutured in place. (See Figs. 4, 5 and 6.)

Third Step. The uterosacral ligaments are brought together posterior to the cervix. The ends are scarified and united with two sutures of chromic catgut No. 2. The ligaments and peritoneum where they now touch are scarified and a suture is taken in the midline and just lateral to the midline.

The first and third steps are done when abnormal conditions exist. The part of attachment in the canal should be sufficiently high to prevent the fundus from doubling backward over the loop and causing a retroflexion of the uterus. The opening in the broad ligament should not constrict the round ligament. The ligaments are sutured taut to offset any stretching that may take place. The muscular (upper) end of the round ligament has the tendency to stretch and break more than the fibrous connection tissue (lower) end; therefore this is taken care of by its use in the canal. All sutures are parallel with the ligaments, thus preventing interference with the blood supply. Each suture goes through the serous layer, ligaments and muscular layer.

ADVANTAGES

This method of operation has the following advantages:

1. It elevates the uterus and holds the fundus anterior.
2. It elevates the ovaries and fallopian tubes.
3. It keeps the uterus in the anterior position by a forward and downward pull of the round ligaments, the resistance of the encircling loop, and the intra-abdominal pressure which is exerted upon its posterior surface.
4. The uterosacral ligaments assist in position by keeping the cervix back.
5. It is noted for its simplicity and ease of performance.
6. There is no chance for adhesions and obstruction.
7. No recurrences have been observed to date.
8. It does not favor postoperative hernia.
9. It does not interfere with pregnancy.

This operation has been used in a large number of cases within the last three years with no recurrences of malposition up to the present time.

SUMMARY

1. There are objections to every kind of operation for uterine malposition at present.
2. Heretofore the term suspension operation has been incorrectly used. It should be reconstruction and correction operation. This is proved when we consider the anatomy and cause of uterine malposition.
3. In doing the first and third step of the operation when certain abnormal conditions exist, are great factors in the success of the second step or Sacher method of operation.
4. The method of operation which I have described overcomes all of the disadvantages and fulfils the cardinal requirements for uterine displacements.

PARESIS & OBSTRUCTION OF JEJUNUM

SECONDARY TO HYSTERECTOMY*

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THE case I wish to report is a patient with paresis and obstruction of the upper 3½ feet of jejunum secondary to a subtotal hysterectomy.

The patient is a female, single, fifty years of age, a public school teacher with a history of feeling a tumor mass in the right lower abdomen for the past fifteen months. The menses are regular. No menorrhagia or metrorrhagia. No vaginal discharge. Normal weight 109 lb. Present weight 105 lb. Pelvic examination reveals a hard, smooth tumor mass the size of a fetal head, filling the entire pelvis and another mass the size of a grapefruit arising from the upper right side of the pelvic mass. A diagnosis of multiple uterine fibroids was made.

A general examination of the head, neck, heart, and lungs was negative. Systolic blood pressure was 110, diastolic 70. Temperature 98.6°F. Pulse 80. Respiration 20. Blood count: R.B.C. 4,960,000, leucocytes 6800, hemoglobin 85, polymorphonuclear cells 60 per cent, lymphocytes 39 per cent, basophiles 1 per cent, coagulation time four minutes. Urine analysis: Sp. gr. 1.020, reaction acid, straw color and cloudy, trace of albumin, leucocytes 5–8 per high power field, erythrocytes fairly numerous, bacteria many.

On June 25 under ether anesthesia a subtotal hysterectomy was performed. The patient was in good condition, pulse between 70 and 90 and temperature not above 101°F. for the first forty-eight hours. On the second postoperative morning there was some distention of the abdomen and that afternoon she vomited once some dark green liquid.

One thousand cubic centimeters of normal saline with 5 per cent glucose twice a day, saline by rectum and nothing by mouth except sips of warm water were the standing orders from day of operation.

A soap suds enema on the second postoperative day brought a great deal of gas but the upper abdomen remained somewhat distended.

Lavage of the stomach brought 75 c.c. of greenish liquid.

Heat to the abdomen was ordered to stimulate peristalsis.

The third postoperative day at 4 P.M. the temperature was 99.4°F., pulse 70, respiration 20. The abdomen was distended, tympanitic, soft on pressure and there was no tenderness. Gas was expelled voluntarily and with soap suds enemas but the abdomen remainded distended and there was nausea and occasional vomiting of a dark green liquid.

A diagnosis of adynamic ileus was made.

The fourth day the same symptoms were present only increased in severity with the addition of colicy pains over the entire abdomen. The abdomen was now distended like a nine months' pregnancy. A soap suds enema brought a lot of gas but reduced the distention very little. Lavage of the stomach twice that day brought up about one pint of brownish liquid that had a sour odor. After lavage of the stomach 2 oz. of castor oil were inserted and returned with the next lavage seven hours later. A soap suds enema did not bring any sign of the oil.

A diagnosis of high intestinal obstruction was made and immediate operation advised. No flat *x*-ray plates were made because the colicy pains were so marked that there was no question of obstruction.

At 8:30 P.M. of the fourth postoperative day the abdomen was opened through the previous incision which was enlarged 2 inches for a better view of the upper abdomen.

A dark red mottled and markedly distended bowel protruded into the incision. This was found to be a distention of the upper 3½ feet of the jejunum and duodenum, produced by a sharp angulation of the bowel and held in place by adhesions for 4 inches along the lateral surface of the distended and collapsed bowel. These adhesions separated without cutting when the distended bowel was elevated.

* Read before Los Angeles Surgical Society October 9, 1931.

The collapsed bowel for about 2 feet distal to the distended jejunum appeared puckered as if in a spastic state. A moderate amount, probably one-half pint, of serum was free in the peritoneal cavity. No flakes or plastic lymph or other evidence of a general peritonitis was seen. The site of operation was clean and no bowel adherent to it.

At a point about 8 inches above the site of obstruction the bowel was incised, a male catheter inserted, the suction apparatus applied and about one quart of light brown thin, watery feces with a great deal of gas removed. Release of tension within the obstructed jejunum caused it to resume its normal color.

The opening was closed with No. 1 chromic catgut and covered with interrupted sutures of linen.

Because the obstruction was so high and the adhesions so delicate we believed it unnecessary to produce a permanent enterostomy or do more to the area formerly adherent. The abdomen was closed without drainage. The next morning the patient complained of crampy pains over the abdomen and had several bowel movements of a light brown color through the rectal tube. There was no distention of the abdomen.

The second day the patient had a yellowish liquid movement with a great deal of mucus. There were occasional crampy pains over the abdomen and she vomited a small amount of greenish liquid. There was no apparent distention of the abdomen.

The third day there was slight nausea and a little distention after a soap suds enema. Lavage of the stomach evacuated a little greenish liquid.

The fourth day the patient was still nauseated. Lavage of the stomach brought about one pint of brownish liquid. The abdomen was more distended. An ampule of surgical pituitrin was given hypodermically. Within about ten minutes she passed a large amount of brownish liquid feces and gas. Most of the distention disappeared but some was still present in the upper abdomen.

A liquid diet, 2 oz. every hour was ordered, in the belief that the sight and taste of food might promote normal peristalsis. Later some milk curds were detected in the stools so apparently something was going through.

Following this up to twenty-three days after the operation for obstruction the patient remained about in this same condition. About one-third of liquids and soft food ingested was taken care of but the remainder was either vomited or washed out of the stomach. From one-half to a pint of yellow or greenish liquid with a sour odor was washed out of the stomach twice a day and the abdomen remained distended like a nine months' pregnancy.

There were no colicky pains and no tenderness over the abdomen. The leucocyte count taken twice during this period was 7100 and 7550 respectively.

A low grade infection with a small Gram negative bacillus occurred in the subcutaneous fat of the wound but this cleared up in a few days and no evidence of peritonitis was seen. The general condition of the patient remained very good as she received 2000 c.c. of normal saline with 5 per cent glucose intravenously or subcutaneously twice a day in addition to the liquids and food she was eating. Nineteen days after the operation for obstruction the blood chemistry showed urea nitrogen 13.2 mg. per 100 c.c. (normal 10–15) and 220 mg. of sodium chloride per 100 c.c. (normal 400–500).

In view of the fact that but slight improvement had occurred after twenty-three days, and with the constant danger of loss of gastric and pancreatic secretion, operation was again advised. This was refused and in spite of consultation with four surgeons during her illness she decided to leave the hospital under the care of another physician. The attending surgeon told me that the condition cleared up entirely without operation in about ten days.

Most of us have had patients with adynamic ileus, obstructive ileus or a combination of the two because one may go into the other. The question we all ask ourselves is what has happened to the unobstructed bowel that is temporarily paralyzed?

Probably the most reasonable cause of adynamic ileus is an over-stimulation of the inhibitory nerves (splanchnics) of the intestines. The irritability of the bowel increases from the duodenum to the ileum, therefore a stimulus of the lower ileum requires a greater stimulus from the duodenum to overcome it.

Normal motion in the bowel is caudad and occurs in the presence or absence of

general peritonitis with direct stimulation, after both vagi and splanchnic nerves are severed. Back pressure is the only force which can prevent the bowel contents from passing toward the ileocecal valve. Stimulation of the lower ileum may create this back pressure.

Wagner in 1922 suggested that lumbar anesthesia might be used to diagnose adynamic ileus from obstructive ileus. Duval in a series of cases found this procedure unreliable. In 16 per cent of mechanical ileus cases an evacuation of the obstructed intestines also occurred.

The principal cause of death which follows high intestinal obstruction is due to chemical changes in the body fluids from loss of gastrointestinal secretions by vomiting and secondarily from circulatory and renal insufficiency.

The blood changes are decrease in the chlorides and a rise in the carbon-dioxide combining power to the plasma (alkalosis). The loss of the chlorides occurs in the vomitus. Dehydration by vomiting occurs only in high intestinal obstruction. Loss of pancreatic juice produces death by dehydration and acidosis. Partial loss of both gastric and pancreatic secretions may be followed by alkalosis or acidosis, mild or severe depending upon the case. Total loss of bile is compatible with life for a long period and produces no marked blood changes.

TREATMENT

The principal treatment for adynamic ileus is: Remove the nervous inhibition by injecting the splanchnics directly with a local anesthetic or indirectly by a spinal anesthetic. The use of choline, atropine and pituitrin is recommended. Intravenous injection of hypertonic saline solution, 3000 c.c. of 3 per cent solution to 50 c.c. of a 25 per cent solution. Food, especially meat and water, stimulates normal peristalsis and is advisable even if returned by vomiting. An enterostomy is often the only safe surgical means of overcoming obstructive ileus but if the obstruction is very high the rapid loss of gastric and pancreatic secretion through the tube may not avert death. In such a case it probably would be well to make a second enterostomy and inject the secretions from the vomitus and upper tube into the lower enterostomy. In all cases sufficient water and saline must be supplied to overcome the rapid dehydration and loss of chlorides.

LEFT FRONTAL LOBE ABSCESS

FOLLOWING DEPRESSED SKULL FRACTURE*

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THE following case represents an example of a compound comminuted depressed left frontal fracture with secondary frontal lobe abscess formation, with recovery.

J. F., aged twenty-four, single, white, male, No. 168,303, was admitted to the Los Angeles General Hospital on July 14, 1931, in a semicomatose condition. A history was obtained that he was in an automobile accident on April 19, at which time he sustained a skull fracture in the left frontal region which was compound and comminuted. He was supposed to have lost considerable brain tissue through the wound. The patient was unconscious for a period of ten hours; was semiconscious for seven days. Ten days after his injury he had an operation for wound repair. He was discharged from the hospital on May 6 (seventeen days after the accident). Several days later he had a vomiting spell and became stuporous, complaining meanwhile of a headache. The region of the left forehead had become swollen and had a greenish aspect. He was taken back to the hospital where the wound was reopened and some necrotic tissue removed. This seemed to relieve the headache and the patient returned to work.

After working about two weeks, he had one spell of headache, associated with vomiting. On the night of July 3, he complained of not feeling well and vomited. He fainted shortly after and was put to bed. The following day he felt some better but was inclined to be very drowsy. The next day he felt irritable and refused to eat. A change in character was noted at this time. He became loud and boisterous, had spells of amnesia and became very profane. This was extremely contrary to his nature. His talk was stated "to have no sense to it." On a few occasions he would drive up to his work, stop the car and doze off to sleep. On awakening he would drive on to his other duties, not realizing that he had done no work. He continued in this condition until July 7, at which time he was ordered back to the hospital by his physician; from there he was referred to the Los Angeles General Hospital on July 14.

Examination on admittance revealed a young adult somewhat stuporous and apathetic. There was a healed scar in the left frontal area. It was thought that a bony defect could be palpated underneath this scar. The pupils reacted to light and accommodation; the right pupil being larger than the left. There was a double choked disc of three to four diopters' elevation. All deep reflexes were increased on the right and a right ankle clonus and Babinski reaction were elicited. A slight right hemiparesis was noted. Impression was that of possible left frontal lobe abscess, secondary to depressed fracture.

X-ray examination revealed a much comminuted fracture with marked depression in the left frontal area of the skull. Laboratory findings were as follows: White blood count 6800; red blood count 4,600,000; polymorphonuclears 75 per cent. Spinal puncture revealed a clear colorless fluid under increased pressure of 400 mm. (water) pressure. There were 20 cells per c.mm. No globulin was present. Urinalysis was entirely negative. The patient's temperature during this time ranged between 98° and 101°F.

Under general anesthesia, an operation was performed on July 20, 1931. A T-shaped flap was turned down revealing a depressed area of bone. Six fragments were removed which were embedded in granulation tissue and cortex. The dura was then opened and a ventricle needle was inserted to a depth of 3.5 cm. At this level a firm resistance was encountered. Puncturing this resistance revealed pus. The needle was withdrawn and a No. 20 catheter was inserted and sewed to the dura, fascia and skin. Usual closure of the flap was made.

The wound drained profusely for about a week postoperatively, and then drainage

* Read before the Los Angeles Surgical Society, October 9, 1931.

ceased. On July 30, the wound was reopened and the tube removed. The abscess cavity was again located with a ventricle needle and the drain replaced. The patient from that time on made an uneventful recovery. The drainage being profuse for the first week. The temperature soon became normal, pulse regular, ranging from 60 to 90. He had no headaches and felt perfectly well. The tube came out of its own accord on August 25. The patient remained symptomless and the wound has ceased drainage.

Eyeground examination showed a markedly subsiding pressure and normal limitations approaching.

Patient was discharged on September 9, 1931.

This case clearly demonstrates the value of *x*-ray examination following injury to the head, before surgical procedures are instituted. It is our policy to elevate all cases of depressed comminuted skull fractures as the fragments are potential sources for brain abscess, as well as other late sequelae. It is also well in this type of case to count the number of fragments revealed in *x*-ray with the number of fragments recovered at time of operation. In this way one is more certain that no remaining fragments are left. If very small, the fragments may be irrigated out with saline solution, by means of a rubber catheter. When the abscess has a firm capsule and is well loculated, prognosis for recovery has been about 50 per cent.

It should always be remembered that when these abscesses are first drained, one should not become alarmed at the absence of drainage material. The abscess has emptied itself; the walls have collapsed, and profuse drainage does not ordinarily ensue until about the third or fourth day. It has always seemed a great temptation to pull or adjust the drainage tubes. It is our policy not to disturb them unless definite indications arise. Dressings are applied daily in these cases and great care is exercised with them.

BLOOD VESSEL LIGATION TECHNIQUE*

the volar aspect of the thumb and is grasped by the adjoining index finger, thus pushing the short end down through the loop. (Fig. 10.)

3. The knot is completed by grasping the short end between right thumb and index finger and both ends are pulled tight, using the left index finger as a directing force to make sure the knot is tight. (Figs. 11 and 12.)

In my experience in teaching students the technique of tying the "granny" and "square knot," I found that the greatest confusion takes place in the steps illustrated, especially in Figures 2 and 8 respectively. It is therefore hoped that the foregoing description and illustrations will help those interested in using these knots in ligating vessels.

* Continued from p. 123.

POSTOPERATIVE HERNIA*

A CONSIDERATION OF ETIOLOGICAL FACTORS

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CHICAGO

IN a review of one's surgical experience, one is often struck by the fact that the occurrence of a postoperative ventral hernia creates a pathological condition which in many instances is more formidable than the pathology for which the primary operation was performed. We have interested ourselves in the experimental production of postoperative hernias in laboratory animals hoping by this study to obtain information that would be of assistance in evaluating the factors responsible for the occurrence of postoperative herniations in clinical work. A correlation of these experimental data with our clinical experience has led us to believe that the most important factors in the production of these hernias may be grouped under the following heads: (1) excessive tension, (2) malocclusion, (3) perforations, (4) infection, (5) muscle paralysis, (6) systemic disease.

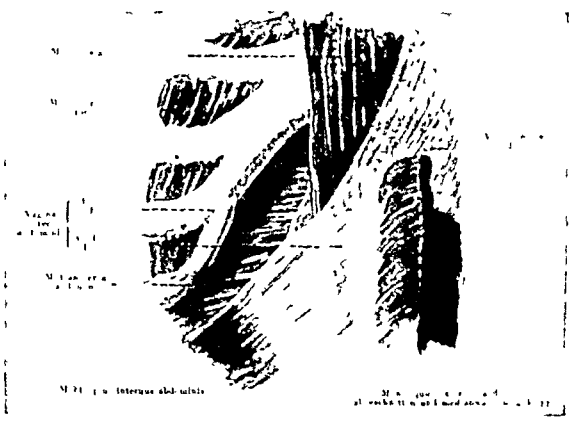


FIG. 1. Transversus muscle beneath posterior sheath of rectus muscle.

EXCESSIVE TENSION

Increased intra-abdominal pressure of an intermittent type such as is caused by coughing, vomiting or straining in defecation or urination is likely to result in loosening or tearing-out of stitches or the forcing of omentum or fat between the approximated layers. The continuous presence of increased intra-abdominal pressure such as occurs in paralytic ileus or incomplete obstruction may also tend to produce the same accidents as occur in the intermittent form and in addition such prolonged distention renders the parietes ischemic and this ischemia markedly delays the healing process.

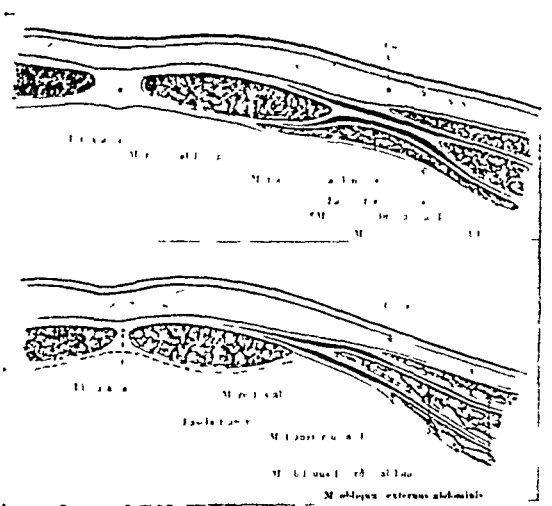


FIG. 2. Sections of abdominal wall illustrating position of transversalis fascia and transversus muscle.

In some instances, excessive tension results from an attempt to bridge too wide a defect with insufficient tissue. There follows a tearing-out of the stitches or the thinning-out of the scar during the process of healing until a weakened area is produced. It has been shown that a simple incision in fascia will be followed by perfect healing provided no undue tension is present. If, however, the incision be sub-

* From the Department of Experimental Surgery, Northwestern University Medical School, Chicago.
Submitted for publication October 14, 1931.

jected to excessive tension, the defect will be bridged with loose areolar tissue similar to that which covers fascia elsewhere.

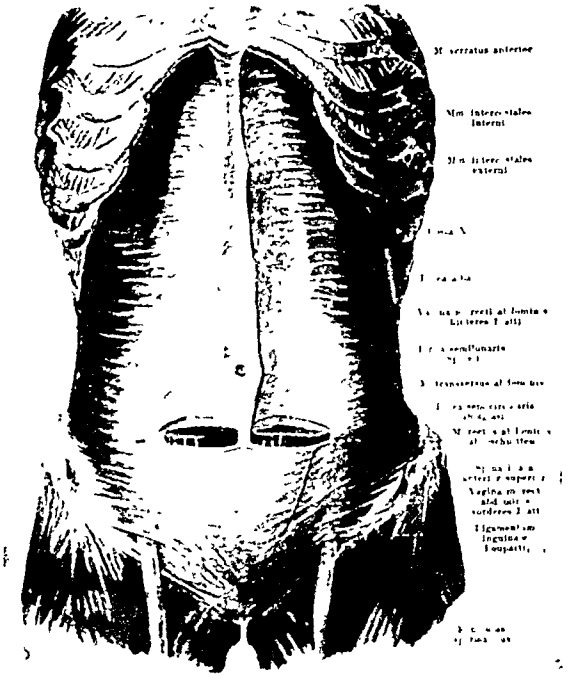


Fig. 3. Transversus muscle.

This areolar tissue will, of course, not offer the same support as normal fibrous tissue.

MALOCCLUSION

Experimentally, it has been shown that like structures will unite most readily and most firmly with like structures, that is, fibrous tissue will heal to fibrous tissue, skin to skin and bone to bone. A capital illustration of this is offered in the usual repair of an indirect hernia in which the conjoined tendon (falx aponeurotica inguinalis) is sutured to Poupart's ligament (ligamentum inguinale). It has been believed that a firm union takes place between these structures but observations on cadavers having herniotomy scars as well as herniotomies requiring re-operation have shown that only a flimsy union occurs and separation of the previously approximated structures takes place. Experimentally, on dogs we have been able to confirm the previous work of Seelig and Chouke, who showed that when the internal oblique muscle is sutured to Poupart's ligament as is usually done in a hernial repair, separation occurs soon afterward. The reason for

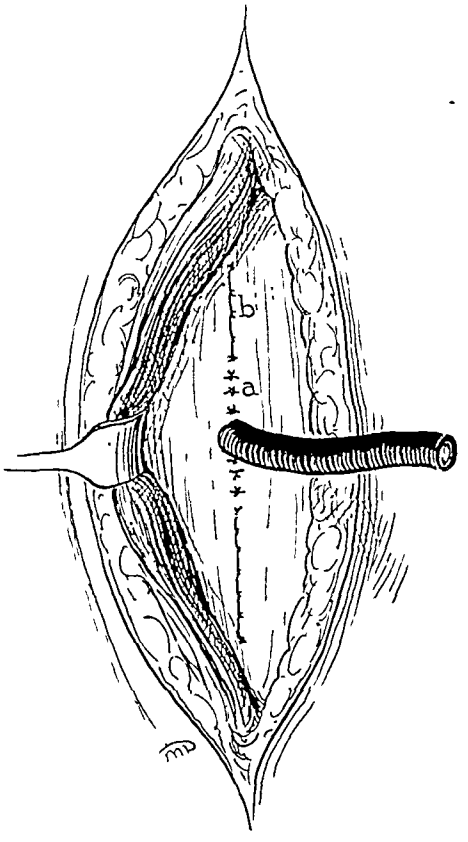


Fig. 4. Use of interrupted sutures above and below a drain.

this is not far to seek. In this procedure, we have the approximation of parallel bands of muscle tissue to parallel fibers of fibrous tissue. The smooth connective tissue investments of the muscle and Poupart's ligament act as a barrier to their common union.

This fact has been taken advantage of by Koontz and others who found it advisable to cut off the edge of the internal oblique muscle and completely remove the smooth covering of areolar tissue that lies upon Poupart's ligament. The approximation of raw surfaces permits a firm union to occur between the muscle and the fibrous ligament. Microscopic examination of sections taken from such united structures shows that the union occurs between the fibrous elements of the muscle, the epimy-

sium, perimysium and endomysium; the muscle tissue itself taking no part in this union.



Fig 5

Figs. 5 and 6. Dog. No. 10

Another elementary fact made note of in the preceding is the removal of the loose areolar tissue that lies upon Poupart's ligament. It has been shown that this loose areolar tissue is a barrier to healing of approximated structures. The reflection of a segment of fascia lata upon itself without the removal of the overlying areolar tissue will not result in firm union of the overlapped surfaces. Complete removal of this areolar tissue with scarification of the opposed surfaces will result in a firm union.

The approximation of the principal fascial planes of the abdominal wall is the first consideration in the avoidance of postoperative ventral hernias. Probably the most important fascial plane of the abdominal wall, judged from this viewpoint, is the transversalis fascia. This layer is of such great importance in our opinion that it would be worth while at this time to review the anatomical relations of this layer.

Our observations on dogs, human surgical material and cadavers have shown that the transversalis fascia is a definite and distinct layer that is easily found and separated in the lower abdomen. It is a fairly thick layer bounded internally by the properitoneal fat and peritoneum and externally by the transversus muscle and



Fig 6.

fascia. As one follows this fascia cephalad, it appears to thin out and finally fuse with the aponeurotic fascia of the transversus muscle. In the upper abdomen toward the midline the transversus muscle and fascia make up in thickness for the diminished thickness and strength of the transversalis fascia. (Fig. 1.)

The transversalis fascia invests the entire abdomen, being continuous with the fascia on the under surface of the diaphragm, the iliac fascia and the lumbar fascia. It is this continuous layer that tends to equalize the intra-abdominal pressure. When a defect arises in this layer any increase in intra-abdominal tension will tend to increase the defect.

In the upper abdomen the transversalis fascia does not appear as a distinct layer since it is fused with the aponeurotic extension of the transversus muscle. The illustration shows that the transversus muscle behind the posterior sheath of the rectus muscle amply supplements the

thinned-out transversalis fascia. In any wound closure, the importance of accurate approximation of these tissues cannot be overemphasized. (Figs. 2 and 3.)

Operative work on the right upper quadrant affords considerable opportunity for the development of postoperative herniation. In many instances, there is a failure to accurately close the transversus muscle and posterior sheath of the rectus muscle. In emaciated individuals all of these structures may well be included in the grasp of one suture. However, in the more muscular or obese individuals, these layers may require at least two layers of suturing to secure approximation of like structures.

In the lower quadrant of the abdomen where the transversalis fascia is usually separated from the peritoneum by a layer of properitoneal fat, it is important in the closure of the wound to approximate the incised edges of the fascia and peritoneum on one side to the like structures on the opposite side. In many instances, retraction of the fascia of one side of the wound often results in a suture of the fascia of the opposite side to a fatty layer. Such suturing results in defective healing. In view of the importance of the approximation of like structures, more attention should be paid to the identification of each layer of the abdominal wall.

PERFORATION

When a drain is permitted to remain in a wound for a prolonged time, healing of the adjacent fascial layers will occur except for the portion held open by the drain. Through this defect a tag of omentum or fat may be drawn on removal of the drain or may find its way into the opening as a result of increased intra-abdominal pressure. Experimentally, it has been shown that the protrusion of a portion of omentum or fat through the transversalis fascia may be the nucleus for the gradual development of a postoperative hernia. In this connection, it may be stated that the increase in size of protruded hernial contents is not only due to the continuous extrusion of omentum and occasionally loops of bowel, but also to the actual growth of the already extruded omental fat.

The avoidance of the prolonged use of drainage material would, of course, diminish the possibility of postoperative herniation in a definite number of cases. In passing, it may be stated that the inadvertant suturing of a drain into a wound with the same suture that is used to close the transversalis fascia and peritoneum may be the basis for a postoperative hernia. Usually, when such suture has occurred, the drain is loosened by removal of the suture. This may loosen the entire approximation of the important layer already noted. Along the tract of every drain there is more or less extension of infection from the surface toward the peritoneum or vice versa, resulting in a premature absorption of those sutures nearest the drain. If such sutures are a part of a continuous suture a serious separation may follow. To avoid this, it is suggested that several interrupted sutures be employed immediately above and below the segment through which any drain penetrates. (Fig. 4.)

Transfixation of a sac in a herniotomy may produce a slit through which a tag of omentum may protrude. Thus, a hernia may develop, the etiology of which is quite different from that of the original hernia. This is especially so in thin-walled, recently developed sacs of the inguinal variety.

INFECTION

Infection plays a definite role in the development of postoperative ventral herniation. Here the cause is not difficult to find since infection causes extensive sloughs of the fascial investments of the abdomen leaving little to maintain the integrity of the anterior abdominal wall. There are several important factors worth noting at this time, which may be of value in diminishing the number of postoperative hernias from this cause. It must be remembered

that of all of the tissues of the body, the most resistant to infection or the most capable of coping with infection is the

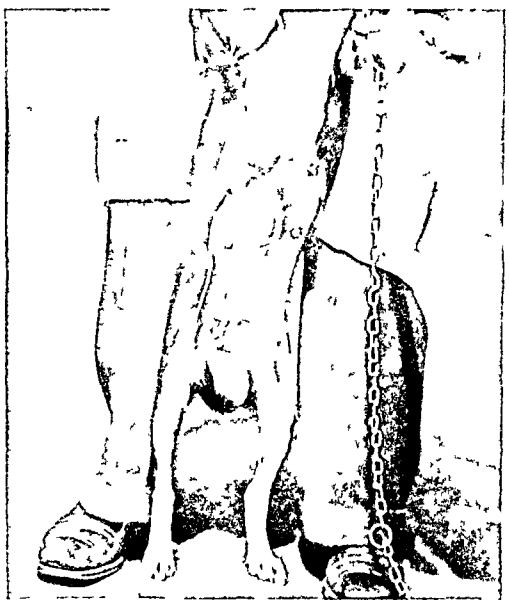


Fig. 7.
Figs. 7–9. Dog No. 15.

peritoneum. Perhaps the least resistant tissues are fat and fascia. When infection is present in the peritoneal cavity good surgical judgment may sometimes dictate the use of drainage material down into the peritoneal cavity. This will tend to carry the infection to such structures as fascia and fat. In a recent paper, Garlock suggested that in suppurative appendicitis, where the McBurney incision is used, no attempt be made to suture the wounds, they being left wide open wherever possible. His comparison with similar cases which were sutured showed that the non-sutured cases were followed by less than half as many postoperative ventral hernias.

It is important in this instance to keep in mind that the opening in layers superficial to the peritoneum must be of such size and patency that there will be no confining of the discharge. The tendency to close fascia and skin snugly around drains leading into the peritoneal cavity is probably one of our commonest surgical errors.

MUSCLE PARALYSIS

This of itself may not be an important factor in the development of postoperative

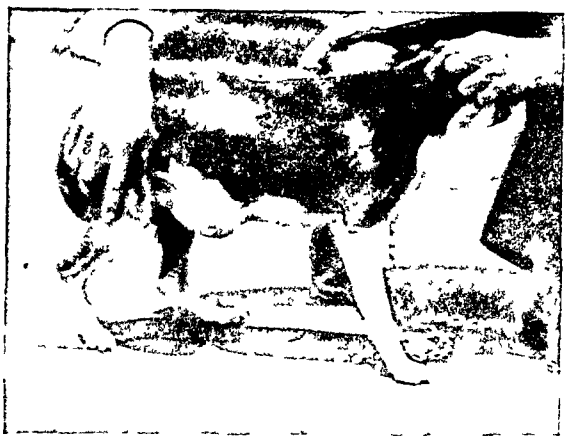


Fig. 8.

herniation but the role muscles play in serving as a splint to a wound by their reflex spasm may influence herniation. In the usual right rectus incision, the medial third of the rectus muscle is separated from its nerve supply, thus producing a weakness in this medial portion of the muscle. Unfortunately, at times as much as half or two-thirds of the muscle is deprived of its nerve supply, thus giving a greater abdominal weakness because of muscle paralysis.

In local infiltration and block anesthesia muscle paralysis also occurs. As a result any undue coughing without the reflex contraction of the abdominal muscles tends to blow out the sutures along the incision, making possible the operation of factors 1 and 2 in the development of a postoperative hernia. This suggests the value of special care in the use of abdominal binders and adhesive strapping so as to supply to the abdominal cavity the necessary support unable to be given by the weakened abdominal wall. Too often, loose application of the adhesive or abdominal binders permits early separation of the approximated structures by the increase of intra-abdominal tension due to coughing or accumulation of gas in the bowels.

One, too, should remember that trauma due to continuous traction on a muscle as occurs when self-retaining retractors are



Fig. 9.

used, produces a temporary paralysis of the muscle. This renders the muscle so deficient in tone that it does not efficiently splint the incised wound in its posterior sheath.

SYSTEMIC CONDITIONS

Diabetes, syphilis, tuberculosis, carcinoma, etc. may interfere with healing sufficiently to prevent firm union of a postoperative wound, thereby favoring the occurrence of herniation.

EXPERIMENTAL CONSIDERATIONS

In our experimental work, we have been impressed with the difficulty of producing ventral hernias in dogs. In many instances, we have excised large segments of both recti muscles in addition to the external oblique and internal oblique muscles and failed to produce any herniation of the anterior abdominal wall. It was this fact which led us to believe that the integrity of the anterior abdominal wall was largely maintained by the intact transversalis fascia and transversus muscle.

Figures 5–9 illustrate hernias in dogs produced in the manner described in the abbreviated accounts of the experiments.

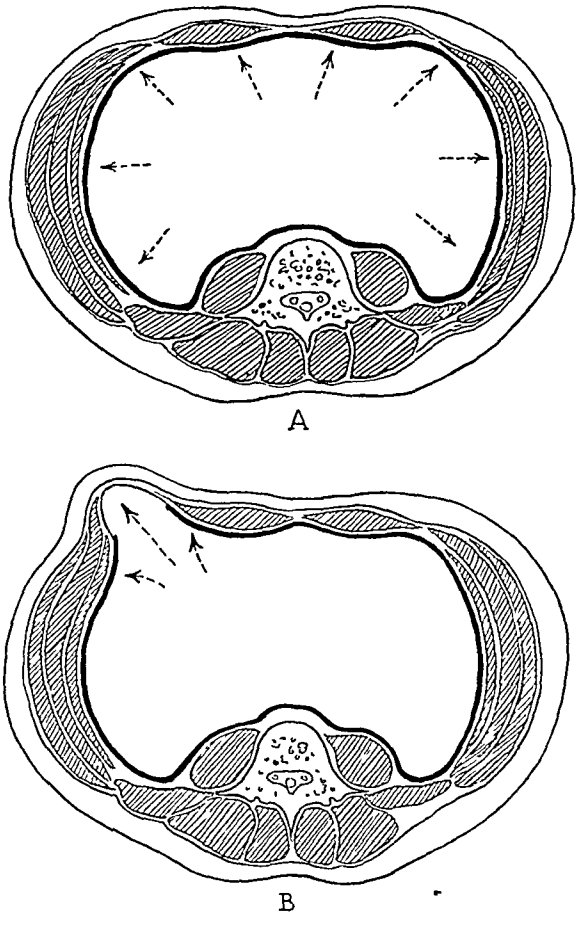


Fig. 10. Distribution of intra-abdominal forces.

Dog No. 10. Importance of an intact transversalis fascia. Figure 5 shows a large ventral hernia produced by removal of all layers of the anterior abdominal wall except the skin and peritoneum to the left of the midline. Figure 6 shows the anterior abdominal wall of this dog with skin dissected away from defects. To the right of the midline is a stellate scar representing a defect in the right rectus muscle This scar is firm and maintains the smooth firm contour of the abdominal wall. To the left is the herniàl sac bulging through the defect produced by the same procedure as employed on the right side with the additional removal of the posterior sheath of the rectus muscle and portions of the transversus muscle. A definite firm ring is present and the extent of the bulge can be appreciated from Figure 5. There was no

defect produced in the peritoneum which remained intact and through which could be seen the subjacent coils of intestines.

In several dogs operated upon in a similar manner, deaths occurred due to strangulation of loops of bowel which herniated through the opening in the transversalis fascia and transversus muscle.

Dog. No. 15. (Figs. 7 and 8). These photographs illustrate the extent and size of a ventral hernia that developed following the protrusion of portions of omentum through a poorly closed peritoneum. This corresponds to the opening left in peritoneal cavities by drains. In the production of this hernia, a portion of the rectus and external and internal oblique muscles was removed (this is not sufficient to produce a hernia). A linear incision was made through the transversus muscle, transversalis fascia and peritoneum. These layers were then sutured in such a manner that omental tags protruded.

Figure 9 shows the same dog reoperated upon three months later. A considerable quantity of omentum had protruded through the anterior abdominal wall defect. This was returned into the abdomen. A firm closure of the peritoneum was made and a fascial transplant was sutured into the defect of the anterior abdominal wall. This resulted in the cure of the hernia.

Dog No. 22. No herniation occurred in this animal following the removal of the anterior and posterior sheaths of the rectus muscle and transversus muscle, leaving the rectus muscle, itself, undisturbed. The defects produced measured 2½ inches in diameter but in two months a firm scar tissue had completely filled the defects produced. The intact rectus muscle was sufficient to maintain the integrity of the anterior abdominal wall.

DISCUSSION

While it is sometimes difficult to reconcile results produced in experimental animals with those in man, there are several points in common which clinical observation will confirm. An individual with a strong untraumatized rectus muscle which has not been denervated is less apt to develop a hernia than the individual whose rectus muscle is flabby, traumatized or denervated during the operative procedure.

In considering defects of the abdominal wall, one may well think of the transversalis fascia and peritoneum as the "inner tube" of the abdomen with the oblique muscles and recti as representing the outer "casing" (Fig. 10). A defect in the inner tube will produce a "bulge" (herniation) because of the unequal distribution of pressure in the abdominal cavity. Usually the immediate cause for the hernia acts soon after the operation is completed, thus allowing structures to insinuate between the incised edges of the parietes acting as an opening wedge which may, as time goes on, cause a gradual increase in size of the hernia.

CONCLUSIONS

From our clinical and experimental observations the following deductions may be made.

1. Postoperative herniations of the anterior abdominal wall are due to one or several of the following causes:

(a) Excessive tension on the suture line.

(b) Malocclusion of the incised layers of the abdominal wall.

(c) Perforation of the wall by fat or omental tags.

(d) Infection of the fascias with subsequent sloughing.

(e) Muscle injury or paralysis.

(f) Systemic conditions such as diabetes, tuberculosis, syphilis and occasionally localized carcinomatous infiltration.

2. The transversalis fascia and the transversus muscle play an important role in maintaining, when intact, the integrity of the anterior abdominal wall.

3. Accurate approximation of like structures is conducive to firm union.

4. The intelligent application of abdominal binders and adhesive tape to the abdomen following closure of an abdominal wound in a manner to relieve the tension on the suture line assists in securing firm union.

PRIMARY TUBERCULOSIS OF THE BREAST*

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THIS patient was a Spanish-American woman, aged twenty, born in Texas, employed as a packer by a canning company in Los Angeles. She has one living child, born at the Los Angeles General Hospital, June 24, 1929 and now thriving normally. The husband died two years ago of pulmonary tuberculosis and the patient has had yearly examinations since then with no evidence ever discovered of tuberculous disease. She presented herself at the Out-Patient Department on July 31, complaining of a sinus in the upper outer quadrant of the right breast which had been in existence for over one year and frequently drained bloody and serous fluid. She stated that she had nursed her baby for five months and that during that period a hard, painless lump developed in the breast. This was opened and drained at the White Memorial Hospital in July, 1930. She states that the wound drained until May, 1931 at which time it closed, only to reopen again in the middle of July, 1931.

Physical examination disclosed a robust, healthy appearing, young woman about 5 feet 3 inches tall and weighing approximately 125 lb., and was essentially negative save for a small draining sinus at the breast margin in the upper outer quadrant. The sinus admitted a fine probe. Palpation revealed a small, hard, firm lump in the breast tissue and apparently attached to the muscle. Firm pressure on this lump caused severe pain which radiated over the right clavicle and down the right arm. When first seen some type of chronic infection or fungus was suspected and smears and cultures were made to rule out actinomycosis, coccidoidal granuloma, etc., and nothing but a few staphylococci were found in the smears or cultures. An *x*-ray of the chest was made to rule out osteomyelitis of a rib and nothing was found. It was then thought we were probably dealing with an infected cyst and cauterization with silver nitrate and other methods were attempted but with no favorable result. The patient was therefore admitted to the surgical ward on August 16, 1931, and operation done on August 17.

The operative procedure was started with a wide infiltration of 0.5 per cent novocaine. The sinus was then injected and to our surprise took more than 12 c.c. of methylene-blue solution. The injection produced very severe pain which radiated down the right arm. The operation was started under local anesthesia, but the patient soon complained rather bitterly and it was completed with gas oxygen. Upon opening the fistulous tract it was found to branch out in several directions through the upper right quadrant of the breast, and to involve the pectoralis major muscle. A small potion of the abscess wall was sent to the surgical pathologist and he reported the presence of giant cells suggestive of tuberculosis. The operation was then completed and every portion of diseased and scar tissue removed. When finished it would have been possible to thrust one's hand into the cavity. A rubber tissue drain was inserted and the wound healed up rather promptly. The pathologist's report on tissue removed was as follows:

"The specimen consists of several irregular tags of skin and subcutaneous tissue to which a small amount of muscle is attached. The specimen is stained with methylene-blue and is said to be tissue removed from a chronic sinus of the breast.

"*Rapid frozen section* reveals chronic inflammatory tissue containing giant cells. Diagnosed as probable tuberculous granulation tissue.

"*Microscopic:* Section showing tubercles and giant cells.

"*Diagnosis:* Tuberculous granulation tissue."

The patient reported at the Out-Patient Department on September 5, with the wound entirely healed and no discharge.

I have taken occasion to review the records of the General Hospital for the past five years and find that 597 patients

* Read before the Los Angeles Surgical Society, October 9, 1931.

were admitted to the hospital wards with some disease of the breast. Of these admissions, 171 were listed as mastitis, 35 as puerperal and 31 as abscess non-puerperal, a total of 237 in all listed as inflammatory. The balance of 360 cases were new growths of which 321 were carcinomatous. In passing it is interesting to note that only one sarcoma of the breast has been observed in five years time and in almost 600 cases. Unfortunately the record system which is otherwise very complete does not list a heading of tuberculosis of the breast, and I am unable to state how many, if any, of these cases should fall in that classification.

Tuberculosis of the breast is a rather rare disease. Deaver reviewed the literature in 1914 and could find only 90 proved cases from 1881 to that date. Since then an average of 2 to 4 have been reported yearly, so there are probably not many more than 150 definitely proved cases in the literature thus far.

The disease can be either primary in the breast or secondary and arising from some other previously existing focus. About 3 out of 5 instances are primary in origin. A typical history would be about as follows: A woman, otherwise healthy, between the ages of twenty and thirty discovers she has a small, slightly painful lump in her breast. This goes on for several months, at the end of which time it breaks down, discharges some caseous material and a persistent sinus forms. If surgery is done at this time and the diseased area entirely eridicated, cure is certain.

The average duration of this disease is about ten months. The treatment is surgery, and the infected tissue should be removed in toto. Some form of inhalation anesthesia should be employed. Following operation the wound almost invariably heals by first intention and the patient experiences no further difficulty. The condition may be, and sometimes is confused with new growths, or with carcinomata. Both tuberculosis and carcinoma can be present in the same breast, but the possibility is so rare that it may be ignored.

TETANUS FOLLOWING ACUTE APPENDICITIS*

understood, but the present-day treatment leaves much to be desired.

It has been suggested that all operative cases involving the appendix and colon be given a prophylactic dose of antitetanus serum. This seems impractical as there would probably be a higher mortality from serum reactions than from the infrequent case of tetanus.

The fact that tetanus bacilli normally inhabit the colon of many individuals is one argument for treating and inverting the appendix stump following appendectomy.

In the treatment of this case the intravenous use of sodium amytal seemed of definite benefit in controlling convulsions.

Some surgeons believe the intraspinal use of the serum to be harmful. The fact that in this case a fatal convulsion occurred ten minutes after an intraspinal injection might tend to favor this theory.

* Continued from p. 99.

TETANUS FOLLOWING ACUTE APPENDICITIS*

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THE advent of tetanus in the postoperative course of an appendix operation is perhaps the most tragic and startling complication with which the surgeon has to deal. Fortunately such an infection is extremely rare, so rare in fact that many surgeons of wide experience have never observed a case.

Six years ago Wainwright collected only 37 cases from the American and British literature, covering a period of twenty years, and since then I have only found 2 additional cases.

Tetanus may occur after any operation, but its occurrence is more likely following operations in which the intestinal tract has been opened, such as appendectomies. This is because the tetanus bacillus normally inhabits the colon of many individuals.

The following case was recently observed at St. Vincent Hospital:

CASE 1. Mr. J. A. S. aged thirty-six, was admitted to the hospital on August 8, 1931. A diagnosis of acute appendicitis was established and an immediate operation advised. At operation considerable free fluid was found in the peritoneal cavity. The cecum and ascending colon were congested and indurated. The appendix was retrocecal and gangrenous throughout. An appendectomy was done; the stump ligated and treated with iodine, but it could not be inverted because of the thick indurated cecum. A Penrose drain was inserted and the wound closed. No cultures or smears were made of the peritoneal fluid.

The pathologic examination of the specimen showed acute gangrenous appendicitis, with a fecolith in the mid portion.

Following the operation the patient developed a diffuse peritonitis and for about eight days his progress was very stormy. Considerable pus drained from the wound and on the tenth day the sloughing fascia allowed the wound to separate. The margins were partially retained by adhesive straps. Gradually the convalescence became smoother but on the morning of the nineteenth day the patient complained of stiffness of his masseter muscles and spasmodic contractures of his abdominal wall. A diagnosis of tetanus was promptly made and treatment started. Two minums of antitetanus serum were given intradermally to test for a possible serum reaction. After thirty minutes no reaction was observed so 20,000 units of the serum were given intravenously and 20,000 units intraspinally. A large dose of sodium amytal (1200 mg.) intravenously effectively controlled the convulsions. Large doses of the serum were given about every four hours; into the veins, the spinal canal, and the muscles; until a total of 120,000 units had been given. For twenty-four hours the patient rested easily and seemed to improve; then convulsions occurred in spite of sodium amytal, magnesium sulphate, and morphine.

Forty hours after the onset of symptoms, 20,000 units of antitetanus serum were given intraspinally. Ten minutes later the patient developed a severe convulsion and expired from respiratory spastic paralysis.

Cultures made from the wound were examined and the material injected into guinea pigs, but no tetanus bacilli could be demonstrated.

Autopsy: The main features of a complete autopsy showed a local peritonitis in the lower right abdomen and a sinus leading from the mesentery of the terminal ileum to the incision.

In the spinal cord there was chromatolysis and actual necrosis evident among the nerve cells of the anterior horn cells. The vessels of the medulla were engorged with blood, in many cases these appeared to be red agglutinated thrombi. The brain tissue showed evidence of early softening in a number of areas.

COMMENT

The bacteriology, pathology, and prophylaxis of tetanus is apparently well

[Concluded on p. 98.]

* Read before the Los Angeles Surgical Society, October 9, 1931.

DO SYMPATHETIC NERVES TRANSMIT PAINFUL IMPULSES?*

REPORT OF CASE

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BROOKLYN, N. Y.

THAT sensory impulses may be carried by the sympathetic nervous system is a conception that is far from proved; however, here is a gradual accumulation of clinical observations which lend support to this idea. Many of these observations have been made on patients who have had somatic nerve pathways of a part interrupted in an attempt to relieve them of pain. With failure to relieve pain by division of the somatic nerves the sympathetic trunks of the part have been interrupted by ganglion extirpation or alcoholic injection with variable results. Other observers have reported the changes following destruction of the sympathetic pathways alone, but since most of this was done by alcoholic injection, the conclusions could be questioned. It is the purpose of this report to record an instance of residual pain in the neck following operative interruption of the somatic nerve pathways of the part.



FIG. 1. Outlined area was completely anesthetic to touch, pinprick, and all degrees of heat and cold. Deep sensibility was absent over left side of neck but present over face and musculature of left shoulder girdle.



FIG. 2. Extent of cutaneous insensibility over upper back and shoulder areas following division of left first, second and third cervical nerves with dorsal roots of fourth cervical segment.

L. I. C. H. No. 30-6589. J. N. aged fifty-eight, a gardener, was admitted on August 14, 1930 to the surgical service, complaining of severe pain in the left side of the face and left lower jaw. The past history was entirely

* Submitted for publication November 4, 1931.

negative except for periodic alcoholic sprees and excessive use of tobacco.

In the early part of 1929 he noticed a small ulcer on the left half of the lower lip which failed to respond to local treatment. In the late spring of the same year he consulted a surgeon who excised the ulcerated area by removing a wedge-shaped piece of the lip. (Section of this tissue showed squamous cell carcinoma.) The wound healed well but during the summer there was a recurrence very similar to the original ulcer. Shortly thereafter he felt several hard, painless lumps beneath the left ramus of the mandible. In September 1929 he was advised to enter the Memorial Hospital of New York for treatment. At this institution the left submaxillary group of lymph glands were removed and radium applied to both the lip and left upper neck area. The growth on the lip completely disappeared. The patient continued with radium treatments throughout October and November 1929, improving all the while in general health. In the early summer of 1930 there was first noticed a dull aching pain beneath the left ramus of the mandible associated with an occasional sharp pain radiating upward over the posterior aspect of the head. Further radium and *x*-ray therapy were of no avail; in fact the pain was made worse by these treatments. Finally after failure of narcotics to relieve the pain which was daily increasing in severity, it was decided to attempt operative relief.

Upon admission examination showed a well-developed but slightly undernourished male of fifty-eight, sitting in bed with head drawn downward and to the left. The left half of the face was moderately swollen, reddish in color and quite firm. Saliva was drooling continuously from the left angle of the mouth which was drawn downward by a firm scar at the site of the original growth. The tongue was swollen and could not be protruded due to its firm attachment by what seemed to be a dense scar in the floor of the mouth. About the operative scar of the neck there was a zone of tissue, not raised, which was of cartilaginous consistency. At the most posterior area of this scar there was a raised area approximately 2 cm. in diameter, bluish domed, very firm and painless to pressure. In fact this entire area was not tender. The patient described his pain as a severe ache in the region of the neck scar with frequent stabbing pain radiating upward both anterior and posterior to the left ear. The remainder of the physical examination was unimportant.

Because of the location of the pain it was thought rational and advisable to divide the upper cervical nerves on the left side, so on August 18, 1930 under ether anesthesia an intradural division of the left first, second and third nerves, and the dorsal roots of the fourth cervical nerve was performed. Following the immediate operative pain he was quite comfortable for one week. At this time he began to complain of a dull ache over the entire left frontal area of his head and occasional sharp pains in the gum of the left mandible. This discomfort increased in severity, becoming agonizing in intensity so that it was decided to divide both roots of the left fifth cranial nerve.

Second operation: On September 8, 1930 under local anesthesia the roots of the left fifth cranial nerve were divided. For two weeks following the second operation he was almost completely relieved of pain. At the end of this period, however, he began to complain of a dull pain in the left side of the neck and temporal region of the head. He described the pain as different from his previous discomfort, likening it to a cramp which increased in severity during any activity such as walking. Examination at this time showed complete cutaneous anesthesia of the entire left side of the head, left half of the neck and left shoulder, yet the patient's discomfort at the end of the two surgical procedures was as great if not greater than at the beginning. Therefore narcotics were again resorted to with partial relief until after six weeks of medicinal confusion the patient died, November 7, 1930.

DISCUSSION

Several observers have advocated the transmission of painful impulses through the sympathetic nervous system, notably Mixter and White, Foerster, Grant, and Fay. The variability of the operative methods used in the reported cases and the inconstancy of the results obtained has left many in doubt. In many instances the nature of the lesion and its exact location was unknown although there was

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every justification to attempt operative relief of the distressing pain.

In the instance herein reported, there were undivided cranial nerves supplying areas adjacent to the point of carcinomatous involvement, but as far as could be determined there was never a complaint which would indicate transmission of the painful impulses over these pathways. Furthermore, all stimuli applied to skin of the left submaxillary region and the left side of the neck as far down as the supraclavicular fossa provoked no response. Bimanual pressure on the carcinomatous mass of the submaxillary region relieved the pain for the moment, only to cause an increase in its severity upon release.

Further experiences with similar lesions has shown that when dividing the cervical nerves a contralateral hemicordotomy should also be performed. If painful impulses pass over the sympathetic fibers of the neck it is almost a certainty that they enter the central nervous system at the first and second dorsal segments. It is therefore more rational to combine the cordotomy with the cervical rhizotomy rather than to perform a second operation on the cervical sympathetic trunk and the carotid plexus.

NON-TRAUMATIC PERFORATION OF COMMON BILE DUCT*

intrahepatic bile duct with escape of bile from the surface of the liver. All of these cases had mechanical obstruction. This condition, as well as the so-called "weeping" gall bladder, would seem to differ etiologically from the one reported here.

Burden[3] has reviewed the work of others on the anatomy of the extrahepatic bile ducts and shown by his own original study that these structures may well be subject to intrinsic pathology. To his findings we may well look for a plausible explanation of pathological rupture. He has shown that the biliary duct wall contains many sacculi into which empty innumerable glands. These glands extend well toward the outer surface of the wall. Normally these sacculi contain no bile, being filled with the mucoid secretion of the glands.

It seems altogether possible for infection to obtain entrance with a resultant abscess or ulceration which finally causes the complete perforation which must have existed in this case.

[3] Burden, V. G. Histologic and pathologic anatomy of hepatic, cystic and common bile ducts. *Ann. Surg.*, 82: 584–597, 1925.

* Continued from p. 103.

NON-TRAUMATIC PERFORATION OF COMMON BILE DUCT*

C. FREMONT VALE, M.D., F.A.C.S., AND HAROLD SHAPIRO, M.D.

DETROIT, MICH.

THE lesion mentioned in the caption is apparently exceedingly uncommon. We were able to find reference to only one other case in the literature; that mentioned by Bailey.[1] The rarity of this acute emergency seems to warrant its report in some detail.

Case No. H-16242. Mrs. C. C. a Mexican girl, aged twenty-three, was admitted to the Medical service about 4:00 A.M. November 15, 1930, with a diagnosis of acute gastritis.

The immediate history was to the effect that she had been taken ill suddenly about 6:00 P.M. the evening before with excruciating pain in the epigastrium, extending through to the back. She vomited once, and nausea with wretching continued throughout the night. The pain increased in severity until it became unbearable.

The past history included several previous attacks of a similar nature during the past four years, though none approached the severity of the present one. Fatty foods seemed to cause some distress, and gaseous eructation afforded some relief. There was never any jaundice or clay-colored stools.

Examination upon admission showed an adult female Mexican of slight stature in good general condition, but restless and evidently acutely ill and suffering much pain. The abdomen was flat with marked tenderness and some rigidity in the upper right quadrant. There was no icterus. Admission temperature 97°F., pulse 96, respirations 22; white blood cells 11,500 with 80 per cent polymorphonuclears. The rigidity rapidly increased as did the pulse and temperature. Operation was advised on the diagnosis of perforation of the gall bladder, possibly of a duodenal ulcer.

Consent could not be obtained until about 10:00 P.M. that evening, twenty-eight hours after onset, at which time the temperature was 102.2°F., pulse 120, 24 respirations; W.B.C. 15,000, 82 per cent polymorphonuclears.

Operation: Spinal anesthesia. Upon opening the abdomen through an upper right rectus incision free clear bile was found filling the subhepatic fossa. This was sponged out but very slowly reappeared. The gall bladder was chronically inflamed with thickened walls and was filled with innumerable small stones (129 were counted). There was no point upon it which leaked bile. The stomach and duodenum were normal. The peritoneum covering the common duct, portal vein and hepatic artery, was edematous, evidently having been dissected up by the presence of escaping bile somewhere beneath it. No stone or point of induration could be felt along the course of the common duct. The gall bladder was opened, the stones removed and since bile flowed freely through the cystic duct a tube was placed in the gall bladder and held by a double purse string suture. A cigarette drain was placed at the foramen of Winslow and the incision closed to drainage.

Postoperative Course: A large amount of glucose and saline intravenously and one direct blood transfusion were necessary to carry the patient over the following five days during which a stubborn peritonitis existed. Recovery then proceeded slowly but smoothly.

COMMENT

The picture of his case reported by Bailey is quite similar to that of ours both as to the immediate acute history and in operative findings. Careful search did not reveal the perforation which was discovered at autopsy to be located posteriorly at the junction of the cystic duct with the common.

Woodruff Smith[2] has collected 11 cases and reported one of his own of rupture of an

[Concluded on p. 102.]

[1] Bailey. Emergency Surgery. 1930. 1: 103.

[2] Smith, Woodruff. Rupture of intrahepatic bile duct with fatal peritonitis. *Ann. Surg.*, 83: 55–68, 1926.

* From Receiving Hospital. Submitted for publication October 14, 1931.

HYDRAULIC VICIOUS CIRCLE AS IT DEVELOPS IN THE INTESTINE

EFFECT OF INTRAINTESTINAL PRESSURE ON THE PATHOLOGY AND PHYSIOLOGY OF THE BOWEL*

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A DISTENDED intestine promptly becomes the site of a hydraulic vicious circle, and the thin wall of the intestine with its poorly protected circulation makes it particularly vulnerable to its strangulating effects. The term "strangulation" has always carried with it the suggestion of blood stasis in the intestine, and it is there that it has been most frequently studied and discussed.

In strangulated hernia, hydraulic vicious circle is a very prominent factor. It is the basis of most of the pathology in the loop of obstructed bowel and is the cause of the rapid production of gangrene which led surgeons, fifty years ago, to say, "Never allow the sun to set on a strangulated hernia." In fact, the whole pathology in a (strangulated) loop of intestine, whether from a hernia or other mechanical cause, is due to the mechanics of hydraulic vicious circle. The initial pressure upon the neck of the hernial sac is very seldom sufficient to stop immediately all circulation into the contents of the sac. Often pain does not develop in a retained hernial loop until several hours after the patient finds himself unable to return it to the abdomen. During this period, the constriction at the ring is not sufficient to produce strangulation. It is, however, tight enough to obstruct the flow of the intestinal contents. The peristaltic action above forces intestinal contents into the loop, but the constriction is so snug that it cannot pass out of it. Thus intestinal contents accumulate until the pressure within the loop establishes a hydraulic vicious circle, with all its consequences.

Once this vicious circle is established, it proceeds exactly as in abscess, and in appendicitis. The material in the loop increases, the effusion increases, and the pressure rises until the circulation in the walls of the intestine is entirely obstructed, and complete strangulation results. The thin wall of the intestine offers little resistance to the closure of these blood vessels.

In abscess, where there are no definite walls, the progress of strangulation is slow; and the walls of the appendix, being thicker, offer much more resistance to the pressure of the accumulating fluid. In the small intestine, on the other hand, the blood vessels having little to support them collapse very quickly. Any gas which may be present in the loop is quickly forced out into the circulation and carried off by the lungs. The effusion always found in the sac of a strangulated hernia is evidence of the pressure upon the walls of the intestine and upon its capillaries, with resulting effusion, from both the outside and inside of the gut. This accounts for the fluid always present in the sac of a strangulated hernia.

Bacteria, by their constant presence in the intestine, play the same role as in the appendix. There is congestion, lack of oxygen, the presence of anaerobes and facultative anaerobes, and infection is very prompt and progresses very rapidly.

The mechanics of obstruction of the intestine in other conditions than hernia is practically the same. When caught under a band of peritoneum it is identical. Volvulus will often produce almost complete strangulation in the same manner. Intussusception has some varying symptoms

* Submitted for publication December 17, 1931.

according to the portion of the alimentary canal involved, but the underlying causes are the same, hydraulic vicious circle, and the results are the same, a gradually developing gangrene.

When an intestinal obstruction develops without producing a loop, as from a band, the hydraulic vicious circle is only partially developed, depending upon the amount of intestine above the obstruction. There will always be more or less interference with the circulation, but if the obstruction is near the stomach, regurgitation will promptly relieve the distention, so that there is but little direct interference with the circulation. For this reason an obstruction in the duodenum will produce copious vomiting, with rapid dehydration of the body; the secretions from the pancreas and liver add enormously to the pouring out of fluid into the intestine, all being expelled by vomiting. But there is less evidence of strangulation here. If this obstruction is low down in the small intestine, the long alimentary tube through which the antiperistalsis must work fails to relieve the intraintestinal pressure, and enough pressure will eventually be produced to cause necrosis and gangrene by the pressure of the distention itself shutting off the circulation.

In every case of intestinal obstruction, interference with the circulation is the most important factor. Whether the toxic symptoms be due to the formation of actual toxins (Whipple, Morton, etc.), or whether they are dependent upon dehydration and depletion of the chlorides, or both, the principal factor in the impending tragedy is the interference with the blood supply through the establishment of a hydraulic vicious circle. The interference with the circulation aids in producing dehydration by effusion into the intestine above the obstruction and together with infection produces necrosis and gangrene which is followed by rupture and peritonitis. Peritonitis following rupture is of course the common cause of death in intestinal obstruction.

Many workers at various times have referred to the obvious disturbance of the circulation and consequent impairment of function occurring in the walls of the hollow viscera as the result of distention from increased intravisceral pressure. Particularly with reference to the intestine and appendix there have appeared during the past few years a number of papers dealing rather specifically with various phases of this problem. It seems, therefore, appropriate to review this subject in an attempt to correlate the various data obtained with reference to the clinical conditions in which such intravisceral pressure may be significant.

In 1907,[1] I made a number of observations on the blood flow in the bowel wall as affected by varying pressures by means of a light within the lumen of the small intestine. It was observed that with distention of the intestine by a pressure of 30 mm. of mercury some capillary streams were arrested; at 60 mm., small veins were arrested, and in most veins the current was slow; at 90 mm., all blood streams were moving slowly, and many, but not all, currents were changing direction frequently; at 130 mm. pressure all circulation ceased, and there was some oscillation of corpuscles but no progress. The following conclusion was made at that time: The demonstration seems complete that distention of the gut (or of other hollow viscera) interfers with the circulation in its wall, and allows infiltration and effusion to take place into its walls and lumen and any other open spaces which may come within its influence. The return circulation is retarded at comparatively slight pressures. Effusion follows, as in all obstructions to venous flow. As the average venous pressure in the intestine under ordinary circumstances probably varies from 4 to 10 mm. of mercury, any pressure beyond that will offer some resistance to the return current.

In 1926, Van Beuren[2] studied the pathologic changes occurring in distended loops of bowel and found that the characteristic

features were areas of hemorrhagic infarction on the antimesenteric surface of the intestine, with varying degrees of necrosis to perforation occurring in these areas depending on the duration of the distention. He stated:

The mechanism of this infarction appears to be as follows: Distention of the intestine increases its diameter. Any increase in its diameter is tripled in its circumferential measurement. In other words, if the diameter of an intestine is increased by distention from 1 cm. to 3 cm., its circumference is at the same time increased from 3 cm. to 9 cm. Thus, the difference in the diameter is only 2 cm., while the difference in the circumference is 6 cm. A moderate increase in diameter, therefore, results in considerable stretching of the wall. The intestinal vessels pass between the layers of the wall along its circumference from their origin at the mesenteric border to their terminal anastomoses at the antimesenteric surface, becoming progressively more thin-walled and more narrow of lumen. Because they are elastic the stretching of the intestinal wall from distention still further thins the vessel walls and narrows the vessel's lumen like a stretched rubber tube. At the same time the pressure from within the intestine tends to flatten out the vessel's lumen. The narrowing of the vessel's lumen and the thinning of the vascular wall are maximum at the antimesenteric surface of the intestine where the terminal anastomoses occurs; and, the distention pressure being constant throughout the lumen of the intestine, the maximum effect is seen at the antimesenteric surface where a union of the three factors of pressure, thin wall and narrow lumen finally results in obliteration of the vessel. This obliteration occurs sooner in the vein than in the artery on account of the less resistant wall. The blood continues to pour through the arterial vessel until the pressure against the obliterated vein suffices to rupture the vessel wall and permit extravasation and coagulation. Finally, the pressure occludes the artery as well as the vein. The area of tissue supplied by these terminal vessels is thus deprived of circulating blood and necrosis occurs. This necrosis is usually first evident in the submucosa and inner muscular coat, but rapidly extends to the other coats of the intestinal wall, and perforation may occur within twenty-four hours after the discoloration due to the hemorrhagic infarct has been first noticed.

In 1927, Gatch, Trusler and Ayers[3] studied the effect of intestinal distention on the circulation of the bowel by recording the effect of such distention on the rate of blood flow in the returning veins. Their observations in general were confirmatory of Van Zwalenburg's, but in addition, afforded an estimate of the degree of circulatory impairment at different levels of pressure. They showed that the blood flow through the bowel wall decreases as the distention increases, and ceases when the pressure within the bowel equals the systolic blood pressure. This result has been confirmed and amplified by Dragstedt, Lang and Millet,[4] who have shown that distention causes the greatest interference with the circulation of the duodenum, and the least with that of the colon. They correlated this variation in susceptibility with the variation in the vascular distribution in different portions of the intestine pointed out by Eisberg.[5]

In 1930, Burget[6] and his co-workers made observations on the pressure developing in closed loops of intestine and noted pressures of 0 to 56 cm. of water. They noted necrosis occurring in the wall of loops distended by pressures of 25 to 30 mm. of mercury.

From the foregoing and many similar observations it is clear that the distention caused by obstruction, etc. can lead to varying degrees of circulatory disturbance in the bowel wall, and if severe to necrosis, ulceration and perforation. The mechanism and the nature of the pathologic changes developing are fairly clear. It is important, however, to consider what functional changes in the bowel wall may occur as the result of distention.

Effect of Distention on Intestinal Motility. It is well recognized that a certain degree of intraenteric pressure will act as a stimulus to peristaltic activity. In excised loops of guinea pig intestine Henderson[7] found pressures of 15 to 25 mm. of water to

be effective. As the pressure is increased, however, motility becomes impaired and finally ceases altogether. The following statement from Alvarez and Hosai[8] expresses the prevailing view:

> Hotz concluded, therefore, that injury to the muscle in peritonitis is due, not to toxins, but to distention by gas. The gas accumulates when absorption is impaired. As Usadel showed, distention of the gut closes not only the arterioles that bring necessary blood to the muscle but also the venules of the portal system, which normally carry the gas away as fast as it is formed. It can easily be seen, then, that once gaseous distention and muscular paralysis appear, they are likely to get worse and worse through the formation of a vicious circle.

Effect of Distention on Intestinal Secretion. Comparatively few observations have been made of the effect of intraenteric pressure on intestinal secretion. Herrin and Meek[9] studied the effect of distention on dogs with varying types of intestinal fistulae. On distending the intestine by means of balloons inflated to a pressure of 80 to 90 mm. of mercury they noted a very marked increase in the secretions from the fistula. As much as 550 c.c. of fluid was formed in a fistula 20 cm. long during the course of twenty-four hours. They state: "Distention is a strong stimulus to intestinal secretion and in obstruction this must work in a vicious circle."

Effect of Distention on Intestinal Absorption. Considerably more interest has been shown in the question of the influence of intraenteric pressure upon the absorption by the intestine of substances that may be present in its lumen. It has long been known that the normal mucosa has the ability of selectively absorbing many nutritive materials and of rejecting or not absorbing other substances not especially different in chemical and physical properties of solubility, permeability, etc. This selective ability of the mucosa, highly specialized as it is, might be presumed to be relatively susceptible to changes of intra-intestinal pressure.

In 1919 Dragstedt, Dragstedt, McClintock and Chase[10] reported a number of experiments which demonstrated that distention of the intestine operated to increase the absorption of toxic materials from the bowel lumen. It was shown that animals could tolerate closed obstructed loops of intestine containing highly toxic material if distention of the loop was counteracted by treating the intestinal mucosa with astringents, and conversely that adding substances like hypertonic magnesium sulphate to such loops which would provoke distention would be followed by intoxication of the usual type. More recently this was demonstrated in a different way by L. R. Dragstedt[11] who prepared closed intestinal loops in such a manner that the lumen of the loop could be aspirated at intervals by a needle thrust through the abdominal wall. In such animals it was observed that the onset of unfavorable symptoms occurred simultaneously with the development of considerable pressure in the loop, and that aspiration of a portion of the loop content, with resultant decrease in pressure, caused a relief in the symptoms. This experiment has been repeated and confirmed by Burget[6] and his co-workers, who have made the additional suggestion that part of the unfavorable symptoms present when the loop is under pressure may be reflex in character.

In 1924 Stone and Firor[12] studied the effect of intra-intestinal pressure on absorption from the lumen of the intestine. The following quotation from their article details their observation:

> Our attention was now turned to the way in which changes of pressure within the lumen of the bowel may affect the absorption of substances present there. It is perhaps well repeated here the observations made by ourselves and others in the behavior of the material found in experimental loops of jejunum. As has been said, this contains a highly toxic element, which when injected intravenously, with proper controls and precautions, causes the death of healthy dogs with a typical syndrome, analogous to that seen in obstruc-

tion. The generally accepted view is that this element is the lethal agent in obstruction, and that its absorption is the cause of death. But if this material be introduced into the lumen of the unobstructed bowel, either by feeding tube or injection through the bowel wall, it is quite innocuous. In other words, it is not absorbed. To throw light on this particular phase, a series of experiments was made with India ink, which may be summarized as follows: (1) Ink was injected into the lumen of the normal intestines exposed by laparotomy, and the wound closed. Such abdomens were reopened at varying intervals afterward and careful search made for any evidence of absorbed ink in the intestinal lymphatics, the mesenteric and retroperitoneal glands, and the liver. None was found. (2) A partial occlusion of the lumen in the duodenum just distal to the papilla of Vater was made by seroserous sutures, kinking the bowel into a sort of sigma shape. The same thing was done in the jejunum about 20 cm. below the first kink. This gave a loop partially, but not completely, obstructed at each end. Into this loop, India ink was again injected. At intervals, the lymphatics, glands, and liver were examined for ink, and none was found. (3) A tie of string (but not thick silk) was placed about the duodenum below the papilla and tied down very hard so as to close completely the lumen. The two sides of the sulcus caused in the bowel walls by this ligature were united by seroserous mattress suture, thus burying the constricting ligature about the bowel. Another hard tie, similarly covered, was placed about the jejunum some 20 cm. below the first. Thus a double complete obstruction and an isolated loop were formed. Into this closed loop was injected again some India ink. We expected these dogs to die. To our surprise some of them survived the initial critical period and seemed to be alright. The abdomen was then opened, and it was found that both ligatures about the bowel had cut through into the lumen and the obstruction was thus relieved spontaneously. The point of greatest interest, however, was that the lymph glands draining the temporarily completely obstructive loop were black with ink, visible both to the microscope and the naked eye.

Comment on this group of experiments will be limited at this time to pointing out that India ink, like the toxins from obstructed loops, is not absorbed when injected into the normal bowel lumen; it is not absorbed when the lumen is narrowed and kinked, but still patent; it is absorbed, however, when complete obstruction exists even for so short a period of time that the animal does not succumb to the obstruction. A possible explanation of these facts might be that pressure within the lumen must reach a certain level before the ink is absorbed through the wall of the bowel, and as long as the lumen was even partially patent, this level was not reached. It is interesting to note also that the dogs which absorbed the ink in these experiments still survived. It may be assumed that they also absorbed toxins, but that the release of the obstruction occurred before a lethal dose was attained, and with the release of obstruction, pressure in the bowel fell, and absorption ceased.

The next series of experiments was performed upon living excised loops of lower duodenum and upper jejunum, obtained from dogs. The preparations were made as follows: The loops were excised without ligation of the stumps of blood vessels or lymphatics leading from the intestine into the mesentery. The mesentery was divided fairly close to the bowel. The loops were suspended in flasks of Ringer's solution, kept at body temperature over a hot water bath, and were discarded for further use as soon as failure of muscle contractions or change of color suggested that the tissues were losing their vitality. With such preparations several types of experiments were carried out: (1) A loop so prepared, closed at both ends by firm ligatures was suspended in the solution for forty-five minutes. At the end of this time the surrounding Ringer's solution was injected intravenously into a healthy dog. No effect of a toxic nature was noticed. (2) A loop so prepared was filled with known toxic content from a dog dying with an isolated obstructed segment of bowel. To one end of this loop a hand-operated pressure bulb was attached by a rubber tube, and the other end of the loop was tied off securely. The preparation was then suspended as in the other experiment, and pressure alternately applied and relaxed, distending the loop with toxin in it in cycles that might be approximately like those of peristalsis. After forty-five minutes the fluid in which the loop was suspended was injected intravenously into a healthy dog and produced death with the typical signs and

symptoms caused by the toxins of obstructed loops of bowel. We interpret these findings to mean that toxin does not pass through a suspended living loop of bowel without some intraintestinal pressure, but does pass when such pressure exists within the lumen.

Gatch[13] has recently reported experiments which seem to clarify many of the apparently contradictory observations on the effect of varying intraintestinal pressures on absorption. Because of the importance of his observations the following quotation from a recent article of his is given:

A ligature is tied about the small intestine, and at a point one to two feet from this ligature the bowel is cut across and a glass cannula is introduced and tied in place so that a segment of bowel is isolated between the cannula and the ligature. The cannula is connected with a pressure apparatus. The blood pressure is recorded. With the segment of bowel outside the abdomen the pressure within it is now elevated till it equals the systolic pressure of the animal. As we have already explained this produces a complete anemia of the bowel. Now we introduce into the bowel a large dose of nicotine or potassium cyanide. Next we gradually lower the pressure within the bowel. There is no sign of absorption of the poison till the pressure reaches about half the systolic pressure. At this degree of pressure there is rapid absorption of the poison, clearly shown by a sudden and great elevation of blood pressure, if nicotine is used, and by a great increase in respiration if potassium cyanide is used. If now the same experiment is repeated except that the bowel, distended to the height of systolic pressure, is replaced within the abdomen, the animal shows immediate signs of absorption. Thus while the distention of the bowel prevents or diminishes direct absorption into the blood stream, it probably increases absorption by way of the peritoneum, because effusion can take place more readily through a thin and damaged bowel wall than through a bowel wall uninjured and of normal thickness. By this method it can be shown that absorption from the intestine varies inversely as distention of the intestine, or stated in other terms, the absorption decreases as the circulation through the bowel decreases, till with the onset of gangrene all absorption must be transperitoneal. From an incarcerated loop or from any loop, the mesenteric vessels of which are obstructed, the absorption must be transperitoneal from the very onset, because here the mesenteric veins of the loop are obstructed very soon. From the free bowel above the site of obstruction the absorption by way of the mesentery will decrease as the distention of the bowel increases. Since observers are pretty well agreed that the toxins from the obstructed bowel are but slowly absorbed through an intact mucosa, and since injury of the mucosa does not occur till a rather high degree of distention has been reached, which would interfere with the circulation of the bowel wall, it follows that the chief route of absorption must be transperitoneal and that the chief absorption of toxins in intestinal obstruction occurs rather late in the course of the disease, after well-marked distention of the bowel has occurred.

To summarize, there are numerous clinical and experimental observations in accord that in conditions of obstruction of the intestine, distention of a sufficient degree to produce alterations in the physiology as well as the pathology of the intestine may occur, that the resulting changes in motility and secretion of the intestine tend to further increase the distention by the establishment of a vicious circle, and that the distention may tremendously alter both the degree and the manner by which substances in the lumen of the intestine may be absorbed.

The bearing that these observations have upon the treatment of the various conditions in which distention may arise is not altogether clear. In case of a simple obstruction seen comparatively early the argument is not complex. There are, however, not a few reports of death, apparently due to toxemia, occurring rather soon after the relief of an obstruction that has existed for sufficient time to cause considerable distention. The earlier opinion that this could be explained by the absorption of toxic material after entering the empty and collapsed bowel below the obstruction

does not seem warranted by the observations previously mentioned, as it appears unlikely that such toxic material will be absorbed by the normal mucosa. On the other hand, as Stone[14] and others have suggested, the relief of the obstruction and the consequent fall in intraintestinal pressure may permit a rapid return of circulation through the bowel wall which may carry off into the general circulation poisonous material which may have penetrated the wall of the intestine during the period when the intestine was distended. This explanation would apparently necessitate the assumption that the transperitoneal absorption presumably occurring during the time of distention is of a less overwhelming nature than that via the mesenteric blood and lymph vessels after the distention is relieved.

It is not altogether an uncommon observation that apparently toxic symptoms may develop after the loops of bowel in the case of a strangulated hernia have been returned to the abdominal cavity from the hernia sac. It is quite possible in the light of Gatch's experiments to suppose that the strangulated loop while in the hernial sac is analogous to a loop of bowel distended while outside of the abdominal cavity to the degree that the only absorption possible is transperitoneal and that the hernial sac has not the absorptive capacity of the free peritoneal cavity. There is thus some justification for the position taken by various surgeons in advising resection of the bowel, which although apparently viable may have been damaged by the distention so that toxic absorption is possible.

There is the alternative of emptying the distended bowel by "stripping" by the method advised by Holden.[15] He places a test tube of considerable size in an incision in the bowel above the site of the obstruction. A ligature holds it in place at the flange. Through this, with the aid of a rubber tube to carry it off, he empties the entire bowel above the obstruction by very gentle stripping with the help of a suitable lubricant.

He reports excellent results. He avoids the shock and trauma of resection and removes the toxins effectively.

It seems probable from the foregoing that most of these problems will eventually be solved by these studies of the circulation in the bowel wall, studies of strangulation and *hydraulic vicious circle.*

Dehydration undoubtedly is the cause of death in many patients. Dehydration from vomiting, from lack of fluid intake, and the dehydration from shock, preoperative, operative, and postoperative, all add to the dehydration. These patients need quarts of salt solution and glucose. Many a patient has died from insufficient hypodermoclysis or intravenous infusion, or even blood transfusion.

Still there remains the positive evidence that the contents of experimentally obstructed loops is a deadly poison when administered to other animals. And it seems evident that many patients have died from the absorption, postoperatively, of this toxin.

The foregoing experiments in absorption with different degrees of pressure in the intestine explain much of the mechanics of this problem.

During the height of the obstruction the intraintestinal pressure, both in the loop and above it, is so high that the veins, capillaries, and lymphatics are all closed, and there can be no absorption. As already shown, when fully distended there can be no absorption from the intestine directly, only transperitonealy. After the obstruction is removed there is gradual relief of pressure, but the sluggish bowel needs some time to rehabilitate itself and be entirely relieved of distention. Congestion, deoxygenation, edema and infection are not immediately removed. In other words enough distention continues for some time to favor more rapid absorption than normal. As already shown moderate distention favors absorption in the presence of comparatively adequate circulation.

This moderate distention favors rapid absorption. The congestion and deoxygenation from poor circulation increase permeability of the capillaries. Complete

distention arrests the absorption of toxins but favors their development and accumulation. The moderate distention which follows the relief of obstruction favors a rapid and overwhelming absorption of the toxins directly from the intestine where they have accumulated. The impaired circulation is at the bottom of all. From this study does not the procedure of Holden[15] seem reasonable and encouraging? By gently emptying the bowel he relieves the circulation, restores normal capillary function more promptly, and removes toxic and infective material. Prompt restoration of adequate circulation is the important result. It is obvious that he must avoid trauma. When carefully done there should be little shock from this procedure.

Meanwhile, all the conditions, circulatory, collapse, shock, toxemia, dehydration, call for copious infusions of fluid hypodermically and intravenously. A well-filled heart has tremendously increased power over a half empty one, and capillaries cannot function properly without an adequate supply of blood and oxygen. Kidneys cannot secrete, nor liver detoxicate when blood pressure falls below a certain height.

We must bring back the blood pressure, and for this, the all-important remedy is an abundance of fluid. Begin before the operation, continue during the operation and then still continue.

Of course peritonitis kills most patients with obstruction, and here again fluid is the trump card.

From these studies we must conclude that both dehydration and toxemia are factors in the cause of the postoperative deaths. When obstruction is high up in, or near, the duodenum there will be a copious outpouring of fluids and chlorides from the liver and pancreas, as well as from the duodenum and stomach by vomiting, while in obstruction in the ileum, or near the colon there will be much less vomiting, but a greater accumulation of toxins and the products of infection. The length of bowel before the stomach is reached will prevent the fluid from reaching the stomach readily, and there will be correspondingly liberal reabsorption of fluid. For the same reason the greater stagnative and circulatory interference will favor formation of toxins. The more extensive bacterial flora, the congestion and deoxygenation are all important factors.

A most instructive study of the mechanics of intestinal obstruction came to me a few years ago in a patient with intestinal obstruction from a large gallstone which had ulcerated into the intestine. This stone, smooth and egg shaped, was just large enough to obstruct the small intestine and still allow the peristalsis to move it down gradually. It had taken approximately a week to reach the ileum where I found it. The distended bowel above it was congested to the point of blueness near the obstruction, but faded to an intense redness a few inches away. The congestion and distention continued upward however, for practically the entire length of the bowel. Near the point of obstruction the intestine was filled with fluid. Near the stomach it was being constantly emptied by regurgitation and vomiting.

The man was ravenously thirsty and evidently dehydrated. He called for water constantly and would promptly vomit all he drank, and seemingly more. He would then immediately call for more water. He had continued thus for several days.

The mechanics were evident. The obstruction was just enough to hold accumulating fluid of secretion, effusion and filtration above it. This distended the bowel to its capacity, stretching the walls and slowing the circulation to an intense congestion. This retarded circulation plus the deoxygenation of congestion increased the capillary permeability (Krog,[16] Landis[17]), allowing an increased outpouring of fluid from the circulation, resulting in the copious vomiting and dehydration.

At the same time it illustrated the fact that the 10 to 15 feet of intestine above this point were sufficient to hold a large quantity of fluid and gas in the distended congested bowel under dangerous pressure, but not sufficient to produce complete strangulation, necrosis or gangrene.

Let us assume that the intraintestinal pressure approached 15 cm. of water. Antiperistalsis would empty some of the contents into the stomach and the pressure would then be lowered to say 8 or 10 cm., just enough to allow capillary circulation with difficulty and great congestion. In a short time the additional accumulation would cause a repetition of the same program.

All the time the peristalsis and the hydraulic pressure were slowly moving the stone onward, steady pressure forcing enough tissues to give way to allow an increase in the diameter of the intestine sufficient to allow the stone to travel slowly downward.

This slow movement of the obstruction relieved the circulation just above it sufficiently to prevent complete strangulation or even infection.

Evidently this area just above the obstruction can withstand stasis up to practical strangulation and arrest for a short time, but if the obstruction is complete as from a peritoneal band a breakdown with necrosis and gangrene results, unless the obstruction is near enough to the stomach to permit antiperistalsis to empty it sufficiently easily to prevent the intraintestinal pressure to reach the point of permanent arrest of circulation in the bowel wall.

Had this stone in its downward movement arrived at a point of bowel sufficiently narrow to arrest it permanently, strangulation, infection and necrosis would have followed.

If on the other hand he could have withstood the dehydration long enough to allow the stone to reach the colon, without finding an impassable stretch, he would have recovered as many other patients of this type have.

In this case incision, easy removal, and copious hypodermoclysis were followed by a prompt recovery.

The mechanics of hydraulic vicious circle were there and beautifully illustrated, but the circle was never completed because one arc of it was movable.

I am proud to acknowledge my indebtedness to Carl Dragstedt for very material assistance in preparing this paper, and to Maurice Visscher for advice, suggestion and encouragement.

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PHLEGMONOUS GASTRITIS*

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GASTRIC phlegmon has been known since 1620 when the circumscribed form of the disease was first noted by Varandaeus. The diffuse form was described in 1861 by Cruveilhier, and since then there have been numerous reports in the literature describing cases of each type. Sundberg, in 1919, published a comprehensive monograph on the subject and included a review of 215 collected cases, and Gerster, in 1927, reviewed an additional 48 cases. During the next four years 12 case reports have appeared and 1 new case is included in this report. In noting the number of cases reported and bearing in mind that the diagnosis of this interesting disease is very difficult and probably quite frequently missed, and that not all diagnosed cases are reported, we find that this subject cannot claim the great rarity attributed to it by most authors. However, the problems of diagnosis and treatment are still unsettled and, for this reason, all cases should be reported. From the increased material, aids to a more accurate and early diagnosis may be had and more satisfactory surgery performed.

We believe that exploratory needle puncture of the upper abdomen with immediate pathological examination of the material obtained and flat plate *x*-rays of the upper abdomen are essential in acute upper abdominal cases where the diagnosis lies



FIG. 1. Autopsy F. A. H. 276. Stomach laid open, showing thickened wall at A. Also flattening of rugae. B. Area from which microscopical sections were cut.



FIG. 2. Autopsy F. A. H. 276. Cross section of thickened stomach wall near pylorus, A. Greater curvature. B. Lesser curvature.

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between perforated ulcer, acute pancreatitis, and gastric phlegmon. It is with the idea of encouraging the wider adoption of these two diagnostic procedures that we offer the following report.

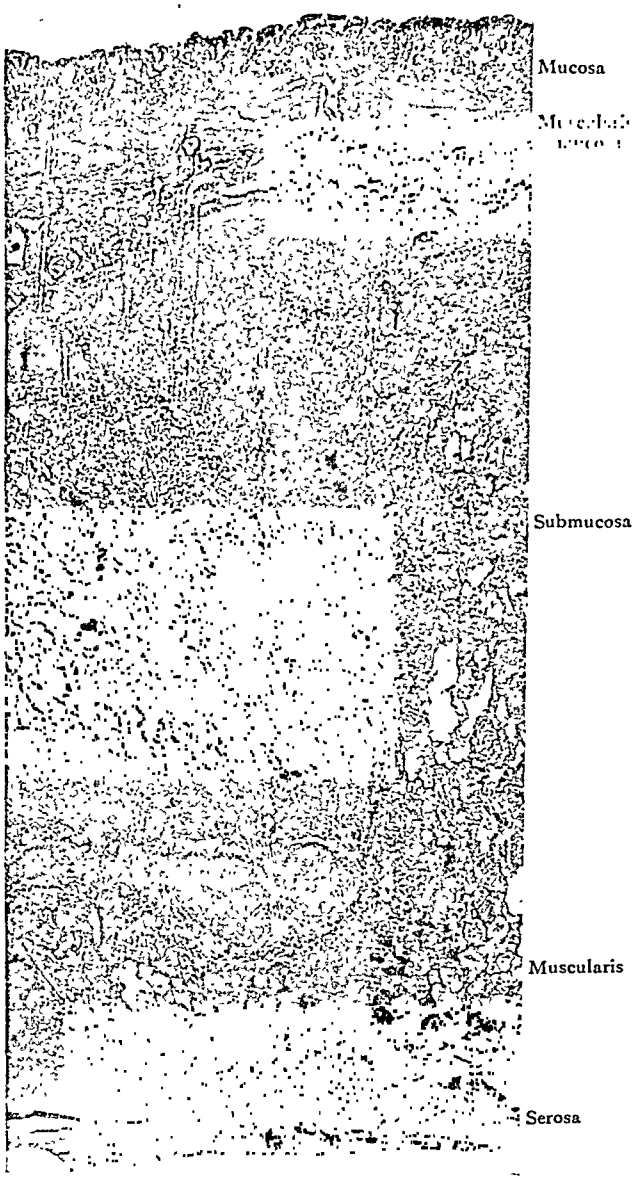


FIG. 3. Autopsy F. A. H. 276. Low power view of section through stomach wall showing various coats. Increased thickness is due for most part to extensive cellular infiltration of submucosa.

Phlegmonous gastritis is a severe inflammatory process of the stomach progressing to purulent infiltration and even necrosis of the stomach wall. There are two very definite forms of the disease: the circumscribed form, and the diffuse form. There may also be a combination of the two. In the circumscribed form there is a definite collection of pus in the stomach wall, in other words, a localized abscess. A typical case of this type was reported by Novak[17] and was cured by a partial gastric resection. The diffuse form is characterized by a more widespread inflammatory process which may include the entire stomach

and extend into the esophagus proximally and the duodenum distally. This form of the disease is twice as common as the circumscribed form and the results following operation are much less satisfactory because diffuse peritonitis is usually a complicating factor. A typical case of the diffuse form is reported in detail in this article.

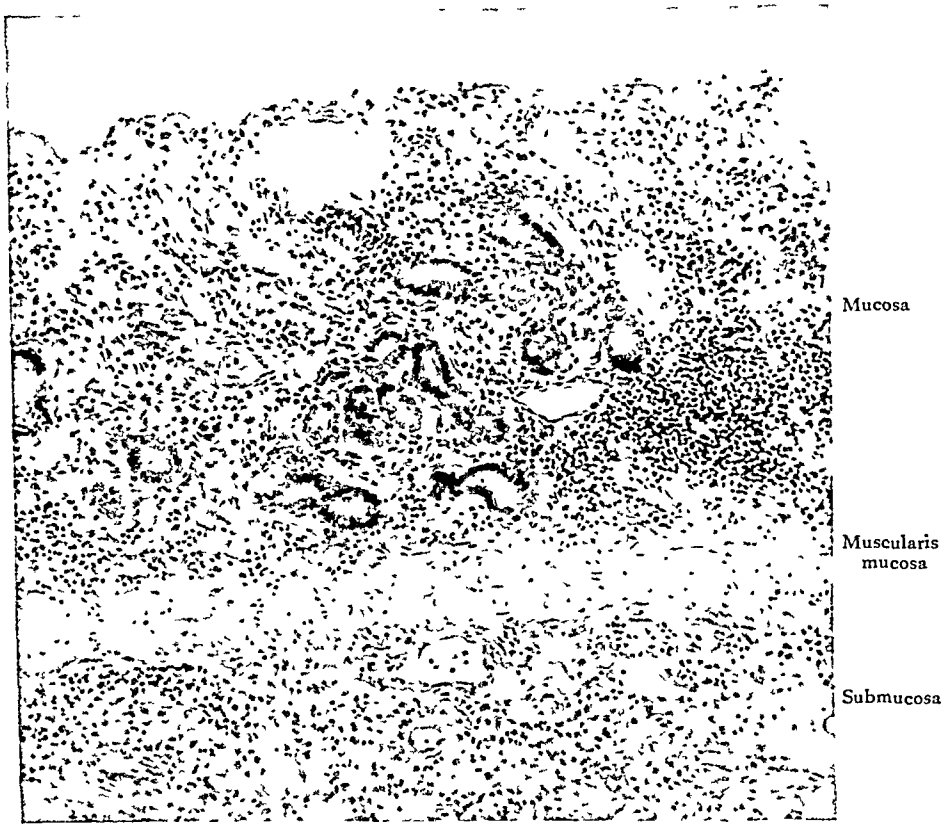


Fig. 4. Autopsy F. A. H. 276. High power view of stomach mucosa with broadened muscularis mucosa and strip of submucosa. Tissues are edematous and infiltrated with polynuclear cells.

As to etiology, much has been written, more has been inferred, and little has been definitely settled. It seems certain, however, that the origin of the inflammatory process may be either local or metastatic. In the first group would be included those cases in which there was present a gastric carcinoma or ulcer and those on which gastric surgery had been done. It has been suggested that gastric phlegmon in a mild form may be not at all an uncommon complication of extensive stomach surgery. Poisons and external trauma are doubtful local causes. In the second group we have the larger number of cases. As no break is found in the mucous membrane the origin of the disease is said to be metastatic, although it has been pointed out that streptococci may penetrate the mucous membrane without causing a local reaction at the point of entry. The inciting bacteria in this group probably enter the submucosa through the blood stream. Included in this group are the cases seen in conjunction with puerperal fever, septicemia, tonsillitis (Brooks and Clinton[3]), erysipelas, and furunculosis (Gerster[8]). Gastric phlegmon has also occurred in connection with smallpox, scarlet fever, and acute polyarthritis. Chronic alcoholism has been thought to be a causative agent and has been noted in about one-fifth of the cases (Albutt and Rolleston[1]). However, there are many cases of alcoholic indiscretion very few of which develop phlegmonous gastritis. Gerster[8]

mentions constitutional predisposing causes such as exhaustion from hard labor, alcoholism, chronic gastritis, hypo-



Fig. 5. Autopsy F. A. H. 276. High power view of submucosa of stomach showing edema and polynuclear infiltration and increased vascularity.

acidity of the gastric juice, and also points out that direct contact with infectious material may be a cause. This latter condition would seem to prevail in cases noted in connection with stomatitis, pharyngitis, purulent bronchitis, drainage of abscesses of the oral pharynx, and extraction of carious teeth. It seems certain that a definite origin for most cases cannot be determined. The streptococcus is the bacteria isolated in most cases (70 per cent), although straphylococci, pneumococci, Bacillus coli, Bacillus subtilis, and Bacillus welchi, have also been noted.

The pathological picture obtained from a study of the post-mortem material is fairly uniform and is exhaustively described in Albutt and Rolleston's "System of Medicine."[1] For a better understanding of the symptoms, course, and results of this usually fatal disease, a few of the main features of the pathological anatomy will be mentioned. The circumscribed form, pathologically, resembles the diffuse form except that the process is more limited. Grossly, the stomach wall is thickened up to eight or nine times its normal size. The stomach capacity may be increased, diminished, or unaltered. The stomach has usually a soft, thick, boggy feel. The serosa may be an angry red or covered with a layer of yellowish fibrin. The mucosa may be hemorrhagic or pale, but is usually smooth with a marked absence of rugae. Microscopically the mucosa shows an abundant round-celled infiltration between the gastric glands and there is

dilatation and cloudy swelling of the blood vessels with extensive degeneration and even necrosis of the secreting cells. The muscularis mucosae may be normal or irregularly swollen and infiltrated with round cells. The submucous coat shows the greatest involvement. It is extremely thickened and edematous and shows immense numbers of leucocytes and an abundant network of fibrin throughout its thickness. The blood vessels are dilated and the tissues of their coats swollen. Microorganisms are present in large numbers. The muscular coat is often very little altered but may occasionally be swollen and edematous. It is less often infiltrated with fibrin or serofibrinous pus. In a number of cases this muscular layer is degenerated, necrosed, or destroyed, allowing pus to reach the serosa. The serous and subserous coats may be unaltered or may show edematous swelling, purulent infiltration, and varying degrees of local peritonitis.



Fig. 6. Autopsy F. A. H. 276. High power view of muscularis (A) and serosa (B) of stomach wall. There is edema and polynuclear infiltration.

The symptoms of this acute abdominal disease are not characteristic. There is usually a history of previous gastric distress followed by the sudden onset of severe upper abdominal pain which is continuous and cutting, and shows a tendency to persist and increase. It is first localized and is increased by pressure in the epigastrium. Later, with the onset of peritonitis, the pain becomes generalized. Vomiting begins early in the disease and continues, becoming more frequent towards the end. Two patients whose cases are reviewed in this report did not vomit. Both were males: one was seventy-two years of age and the other a carcinomatous patient of sixty-two. Old age and debility may account for the absence of vomiting in these 2 cases. The vomitus consists first of a watery, mucoid material which later becomes bile-tinged

and then greenish. Frank pus has been reported. Fever may be absent, but the temperature is usually 100 to 104°F. The



FIG. 7. Preoperative *x*-ray.

pulse is at first full and strong, but later becomes thin and rapid. There is increased resistance in the epigastrium. Deininger (1879) noted a relief of pain when the patient was made to sit up. The patient appears acutely ill and is restless and anxious, later becoming dehydrated, and finally going into delirium and collapse. The white blood count is usually high: 20,000 to 30,000, with a correspondingly high polymorphonuclear count.

From a glance at the symptoms of this acute abdominal condition, it is clear that the diagnosis will be a difficult one to make because of the lack of any definite diagnostic sign or symptom. For this reason it is very rarely made before operation or necropsy. A perforated gastric or duodenal ulcer is most often suspected and acute pancreatitis is also a frequent preoperative diagnosis. Neuhoff[18] reports a case in which an abdominal puncture was done and a considerable amount of cloudy fluid containing streptococci was obtained. A similar procedure was done in our case and a thin, turbid, slightly reddish fluid was obtained. A typical history together with streptococci in a turbid fluid removed by abdominal puncture should lead one to suspect a gastric phlegmon, and would tend to rule out acute pancreatitis and perforated ulcer. It would seem logical to expect a very thick walled stomach to produce an area of increased density on an *x*-ray film, but the film taken of the case reported in this article was only suggestive of a thickened stomach wall. As far as I know, there are no other cases reported where *x*-rays were taken during the acute stage of the disease, and I would urge this procedure in all suspected cases. High fever and frequent vomiting of bile-stained watery material together with relief from pain on sitting up (Deininger) speak for gastric phlegmon as against perforated ulcer. A preoperative *x*-ray of the abdomen might give definite evidence of a perforated viscus, and a review of the films in cases which at operation proved to be phlegmonous gastritis might lend some clue to the *x*-ray diagnosis of this condition. With the abdomen open the diagnosis is often made or confirmed by aspirating pus from the submucous layer of the stomach wall.

The prognosis is grave. Death is the usual outcome in the extensive diffuse type. In the localized type the abscess may rupture into the stomach and a spontaneous recovery ensue or the involved portion of the stomach may be resected giving the case a better prognosis. Cases which have recovered after simple drainage of the abdominal cavity must be classed as spontaneous. Perhaps many others have recovered which were never diagnosed.

The most consistent complication and the immediate cause of death in most cases is a diffuse peritonitis.

The treatment is surgical, but the procedure carried out at operation will depend upon the type and extent of the disease. Gerster[8] reviewed 18 cases in which gastric resection was done. Of these, 8 patients

recovered (44 per cent), so that it seems clear that gastric resection for the circumscribed type of gastric phlegmon is the treatment of choice. In the extensive diffuse type, a resection would be futile, and in these cases a palliative jejunostomy and abdominal drainage seem advisable. Multiple incisions in the stomach wall down to the mucosa with adequate drainage has been advocated. Gastrostomy and gastroenterostomy have been done with very little reason or success. It has been suggested (Weinstein and Klein[28]) that the entire stomach be brought out of the abdomen, as in a Mikulicz operation for malignancy of the colon, and multiple incisions made through the serosa and submucosa for drainage and a jejunostomy done for feeding purposes. This procedure has never been done, but would seem logical for otherwise hopeless cases of extensive diffuse phlegmonous infiltration of the stomach wall. In our case the disease was very extensive, extending into the duodenum and esophagus and involving the entire stomach. A palliative jejunostomy was done and the peritoneal cavity drained.

CASE REPORT

The following is a summary of the history and findings in our case which is No. 12 in the table of reviewed cases:

Miss A. M., an Irish housekeeper forty-eight years of age was admitted to the Fifth Avenue Hospital on the service of Dr. F. W. Bancroft at 12.40 P.M., March 19, 1930. She was complaining of pain in the abdomen together with vomiting of six days' duration.

History. Symptoms began with a sudden sharp pain in the epigastrium. This pain rapidly shifted to the right lower quadrant and she began to vomit. Then the pain spread to the entire abdomen and the vomiting continued up to the time of admission. Her bowels had been moving regularly and she gave no history of previous indigestion or vomiting. Her previous history was essentially negative. She had not lost weight and gave no history of excessive use of alcohol. Her menstrual history was entirely normal.

Physical examination revealed an acutely and gravely ill woman lying quietly in bed and giving the typical picture of a generalized peritonitis. Her abdomen was slightly distended and rigid throughout. There was marked spasm of both rectus muscles, rebound tenderness throughout and definite skin hyperesthesia. The abdomen was typanitic except for dullness on percussion in the left upper quadrant. Rectal examination revealed tenderness in both fornices and the cervix was very tender on manipulation. There was no obliteration of the liver dullness. Flat plate *x*-ray of the abdomen did not show a definite mass or any evidence of intestinal obstruction.

Laboratory Data. Her temperature was 103°F., pulse 120, and respirations 24. Blood count: White blood cells 24,500, polymorphonuclears 96 per cent, lymphocytes 4 per cent, and non-lobulated polymorpho-nuclears 86 per cent. Coagulation time four minutes. Urinalysis: Acid reaction, trace of albumin, no sugar, occasional finely granular cast, occasional red blood cells, and a moderate number of white blood cells.

The case was seen by several of the attending surgeons. All agreed that the patient had a generalized peritonitis and each suggested a possible cause for it such as acute suppurative appendicitis, perforated duodenal ulcer, acute pancreatitis, etc. An exploratory laparotomy was definitely indicated from the findings and was performed immediately.

Operation. Before making an incision a lumbar tap needle was inserted into the left upper quadrant and about 1 cm. of turbid fluid was removed from the peritoneal cavity. A mid-paramedian incision was then made. Diffuse peritonitis was seen and a large amount of turbid fluid mixed with flakes of fibrin was evacuated. The appendix was first sought for and drawn up into the wound. The vessels seemed to be congested, but there was no evidence of any primary trouble there and its removal was considered inadvisable. The gall bladder was palpated and found to be normal. The duodenum was normal on palpation and no thickening of the pancreas was felt. The stomach from the pylorus almost to the cardia felt about 3 cm. in thickness. This was the only lesion that could be seen during the exploration and suggested a phlegmonous gastritis. There was no evidence of fluid in the lesser sac. It was thought advisable to do a jejunostomy in

order to relieve paralytic ileus and vomiting. This was done by inserting a No. 18 French catheter into the jejunum through a purse-string suture and inverting, as in a Witzel gastrostomy. The tube was brought out through the omentum and then through a lateral stab wound. One cigarette drain was placed through Morison's space and one lateral to the ascending colon. Two drains were placed to the right lumbar gutter, and two cigarette drains brought out at the upper and the lower angles of the incision. The peritoneum was closed and the wound loosely united with tension dermal sutures over buttons and packed with vaseline gauze.

Progress. Following the operation the patient's temperature steadily rose and twenty-four hours later it was 106°F., spiking down to 102°F. at mid-day. The following morning it reached 106.6°F. She was given saline therapy and was able to take some water by mouth without vomiting, but she presented a flushed face, bright eyes, cold extremities, rapid respirations, and the appearance of a doomed patient. She died three days postoperative.

The following is a report of the post-mortem examination performed by Dr. D. S. D. Jessup on March 22, 1930:

The body is that of a woman of about fifty, of medium frame and abundant adipose. To the right of the navel there is a vertical operative wound, 20 cm. long which is open down to the peritoneum. There is thin pus welling out from the bottom of the wound through openings of the peritoneum which occurs at several points. To the left of the navel there is a small opening with a drainage tube leading down through the omentum. The loops of the small intestine are matted together by a fresh exudate of lymph forming light adhesions. The appendix lies free and does not appear inflamed. The peritonitis extends through the whole cavity and over the surface of the liver.

The uterus is moderate in size. The tubes and ovaries are small and covered with exudate of lymph. The spleen is enlarged and soft with exudate on the surface. The heart is normal in size and the pulmonary valves are normal. The tricuspid admits three fingers. There is slight thickening along the edge of the mitral cusps. The aortic cusps are normal and near the cusps there is atheromata of the aorta. The coronary arteries appear normal. The heart muscle is normal. The left lung shows some fresh exudate of lymph over the lower lobe with marked congestion, and the upper lobe shows slight scarring at the apex. The right lung shows a large amount of fibrinous exudate over the surface. The liver is large, rather pale, and very friable on section. The pyloric end of the stomach shows increased thickness of the muscularis. The mucosa of the stomach is markedly injected with small areas of hemorrhage, but there is no break in the wall. The thickening is a diffuse process and on section there is an exudate of purulent fluid from the cut surface. The pancreas is normal in appearance. The small intestine is normal except for a few areas of congestion and the large intestine contains fecal matter in the upper portion, mucous in the lower portion, and also shows small areas of congestion. Both kidneys are normal in size, the markings are quite distinct, and the capsules strip quite readily. The adrenals show some congestion. The uterine canal is normal, the muscularis is very fibrous, and there is a small fibroid of the wall.

Bacteriological Examination. Smear of the lung shows many intracellular streptococci and a smear of the liver shows fewer streptococci.

Cultures taken of the upper surface of the liver, upper surface of the stomach, left quadrant, right quadrant, pericardial sac, and the heart muscle show gram positive bacilli, other slender gram positive rods (diphtheroids), and streptococci (?) not hemolytic. Aseptic cultures taken of seared spleen show hemolytic streptococci. Blood cultures were not done.

Anatomical Diagnoses:

1. General purulent peritonitis.
2. Acute fibrino-purulent pleurisy.
3. Congestion of the lungs.
4. Phlegmonous gastritis.

Microscopical Examination. The lungs at one point show marked congestion and edema. The pleura is thickened and covered by a positive fibrin mixed with polymorphonuclears. The underlying lungs show congestion. The heart muscle shows separation of the fibers by connective tissue growth. The aorta shows basic degeneration. There is fatty infiltration and congestion of the liver and old blood pigment, also some polymorphonuclear infiltration in Glisson's capsule. The kidneys are congested and there are inflammatory changes in some of the tufts. The pancreas shows some

Case No.	Sex	Age	Occupation	Probable Etiology	Prominent Symptoms	W B. C.	Preop. Diagnosis	Operative Procedure	Autopsy	Bacteriology	Result	Author
1	M	72		Followed operation for excision of ruptured duodenal ulcer	Sudden severe pain in upper abdomen. Abd rigidity. No vomiting	17,300	Perforated ulcer	Excision of a perforated ulcer of the duodenum	Pulmonary edema and congestion. Phlegmonous gastritis. Peritonitis	B. welchi	Died	Morton & Stabins
2	M	19		Developed during a streptococcus pneumonia	Symptoms of pneumonia plus tenderness in epigastrium	22,000			Lobar pneumonia. Phlegmonous gastritis	Lungs: Hem. streptococci. Stomach: Hem. streptococci	Died	Meyer, Brams, & Guy. Case No. 1
3	M	42		Carcinoma of stomach. Bilateral pneumonia.	8 months gastric distress. Tender palpable mass in epigastrium	30,000		Operation refused	Bilateral pneumonia Diffuse phlegmonous gastritis	Not done	Died	Meyer, Brams, & Guy. Case No. 2
4	M	37	Clerk	Alcoholism. Ruptured gastric ulcer	Acute abdominal pain, vomiting and shock		Perforated ulcer	Closure of perforation. Witzel type, jejunostomy	Peritonitis at operation		Recovered	Weinstein & Klein
5	F	62		Idiopathic	Severe abd. pain, vomiting, and diarrhea. Temp. normal	25,000			Phlegmonous gastritis	Streptococci and B. proteus	Died	Robb
6	M	62	Laborer	Carcinoma of stomach	4 yrs. epigastric distress. Acute severe abd. pain 2 days. No vomiting. Board-like abdominal rigidity		Perforated ulcer	Exploratory Drainage	Phlegmonous gastritis. Peritonitis. Carcinoma of stomach	Streptococci	Died	Schaefer
7	F	21	Dressmaker	Idiopathic	One year ulcer history. 3 days acute epigastric pain		Covered perforation of stomach	Invagination of a 3 cm. necrotic area of ant. stomach wall & gastroenterostomy			Recovered	Paugger. Case No. 1
8	M	18	Locksmith	Chloride of zinc	Loss of appetite and pain 2 hrs. after eating. Sudden abd. pain, vomiting & chill		Covered perforation of stomach	Exploratory. Drainage	Peritonitis at operation		Recovered	Paugger. Case No. 2
9	F	43	. .	Carcinoma of stomach	5 yrs. dull epigastric pain. Sudden acute abd. pain 6 days after roentgen examination		Covered perforation of gastric carcinoma	Partial resection. Braum anastomosis	Peritonitis. No phleg. gastritis. Avertin necrosis of rectal wall Resected spec. showed phleg. gas		Died 27 days P.O.	Paugger. Case No. 3
10	F	28	Housemaid	Carcinoma of stomach	6 mo. gastric discomfort then sudden severe abd. pain & vomiting. Tender swelling in epigastrium. Fever		Inflammatory tumor of omentum with covered perforation	Billroth 1 resection	Op. specimen: Ca. & phleg. gastritis P. M.: Peritonitis & encapsulated abscess	. .	Died	Paugger. Case No. 4
11	M	18		Partial starvation	. . .	. .	. .		Congested spleen. Diffuse phleg. gastritis		Died	Mongin
12	F	48	Housekeeper	Idiopathic	Sudden sharp abd. pain & vomiting. Temp. 103°F. Pulse 120	24,500	Generalized peritonitis	Jejunostomy and drainage	Diffuse phleg. gastritis. Peritonitis	Hem. streptococci. B. Welchi & nonhem. streptococci	Died	Author
13	F	56		Idiopathic	Vomiting at meal time for 20 yrs. Attacks of upper abd. pain with vomiting & fever	7,900	Chronic cholecystitis & cholelithiasis	Partial gastric resection & posterior polya anastamosis	Cellulitis of thigh. Lung infarcts & abscesses Phleg. gastritis. Peritonitis	. .	Died 20 days P.O.	Rankin & Miller

increase of stroma with edema. The adrenals show congestion and some round cell infiltration in the medulla. There is a fibrosis of the uterine wall and the endometrium is atrophic.

The esophagus near the stomach shows some edema of the stroma. The stomach glands extend up for a considerable distance and are covered by squamous epithelium for quite an area. The marked thickening of the wall of the stomach is caused by acute inflammatory exudate into the submucosa, this being made up of polymorphonuclear cells with fibrin and areas of necrosis. The inflammatory process goes through the muscularis where the muscle fibers are separated by edema and where there are many large mononuclear cells, these appear to be either monocytes or phagocytes. The serosa is involved in the inflammatory change and the superficial layer of the mucosa lining the stomach has the appearance of granulation tissue with many small thin-walled blood vessels. The spleen is markedly congested.

Microscopical Diagnoses:

1. Fibrino-purulent pleurisy.
2. Phlegmonous gastritis.
3. Streptococcus peritonitis.
4. Acute hepatitis.
5. Myocarditis.

SUMMARY

Of the 13 cases reviewed 9 patients were operated on and 3 recovered. In each case of recovery the disease was of the localized type and in one of these the diagnosis was made on inspection only. In 6 of the cases (46 per cent) there was an associated gastric carcinoma or gastric or duodenal ulcer. One case was precipitated by the ingestion of zinc chloride and another developed during the course of a streptococcus pneumonia. Starvation plus the eating of heavy rough grass is said to have caused a fatal phlegmon of the stomach (Mongin[13]). Seven of the patients gave a previous history of gastric distress. In 7 of the 9 patients operated upon a preoperative diagnosis of perforated ulcer was made. In one of the remaining cases a diagnosis of general peritonitis was made and in the other cholelithiases was the diagnosis. In 5 cases bacteriological studies were done and 4 showed streptococci and one Bacillus welchii. In 1 case the streptococcus was found in association with Bacillus proteus and in another with Bacillus welchii.

CONCLUSIONS

1. Phlegmonous gastritis is not as rare a condition as it was formerly thought to be. There are two forms of the disease, the diffuse and the circumscribed. The diffuse form is fatal; the circumscribed form is less so.

2. The diagnosis has never been made before operation or autopsy.

3. Abdominal puncture after the technique of Neuhoff[18] and the routine preoperative *x*-rays of the abdomen are suggested as additional aids to diagnosis.

4. Treatment must be surgical, the procedure elected depending on the type of the disease. Localized type: gastric resection; diffuse type: surgery offers the palliative relief of a jejunostomy.

5. The etiology is not clear, but in a large number of cases there is preexistent stomach pathology, often carcinoma or ulcer, which may provide for the easy entrance into the submucosa of the streptococcus, the organism usually isolated from the phlegmonous gastric wall.

6. All cases should be reported completely so that with increased material at hand the symptoms and diagnosis may be more clearly understood and the treatment improved. The surgeon when confronted with an acute upper abdominal inflammatory lesion should always consider the possibility of a gastric phlegmon.

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[For Remainder of References see p. 50.]

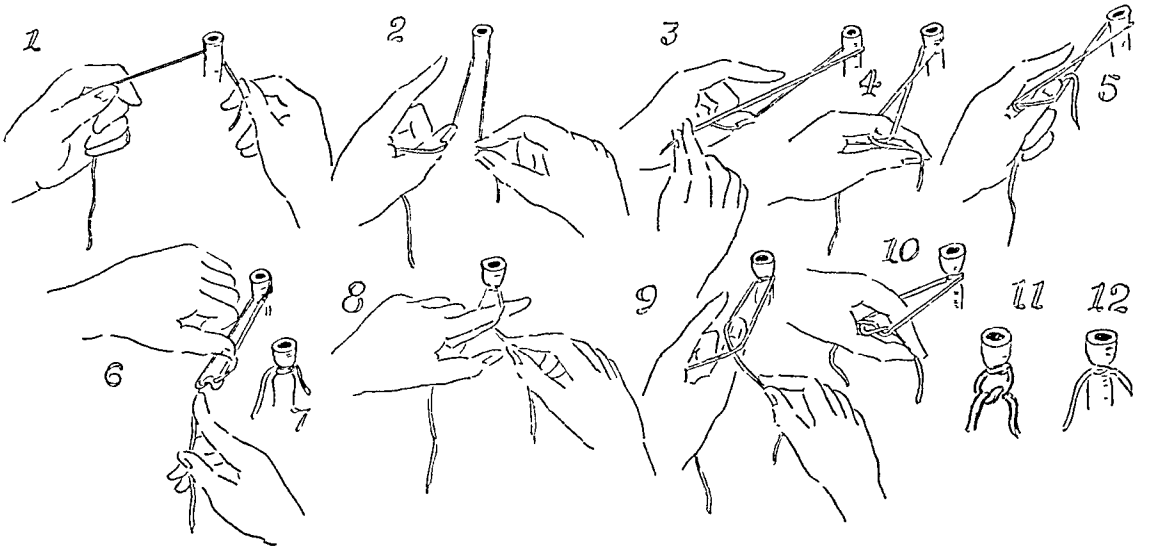
BLOOD VESSEL LIGATION TECHNIQUE

SUCCESSIVE STEPS IN TYING THE "GRANNY" AND "SQUARE KNOTS"*

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THE technique of ligating blood vessels has become so refined and so standardized that perhaps one should offer an apology for discussing its details, but there are certain points in its performance which if kept constantly in mind by the surgeon, will often avoid serious accidents during and after an operation.



FIGS. 1–12.

1. The long end of the ligature is held in the palm of the left hand with the short end projecting between the thumb and index finger.

2. The short end of the ligature is brought around the vessel to be ligated with the right hand and is held between the tips of the thumb and index finger. (Fig. 1.)

3. With the left taut part of the ligature hugging the inner and volar part of the thumb of the left hand, the short end of the ligature is brought over to the apex of the angle between the thumb and index finger of the left hand. (Figs. 2 and 3.)

4. The left index finger is inserted into the loop thus formed, and the short end is crossed over the palmar surface of same towards the tip and supported by palmar surface of the adjacent thumb and brought up through the same loop. (Figs. 4 and 5.)

5. The short end is grasped by the right thumb and index finger and the first tie is completed by crossing the long end of the ligature over the short end, thus avoiding undue friction and breaking the ligature. (Figs. 6 and 7.)

To complete the "granny knot," the above steps are repeated in the same order.

To make a "square knot," at this stage, the long end of the ligature is brought over the outer and posterior part of the left index finger and the short end is brought across on the palmar surface of the index finger, thus leaving the latter in the loop produced, pointing downwards. (Fig. 8.)

1. The adjoining thumb pushes the index finger out of the loop and takes its place. (Fig. 9.)

2. The short end is then brought across

[Concluded on p. 89.]

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A MULTIPLE FILM CASSETTE

WITH A GRATING LOCALIZER TO AID IN A MORE PRECISE IDENTIFICATION AND LOCALIZATION OF RENAL CALCULI AT OPERATION*

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A LOCAL kidney film at operation has been used for years by urologists for the double purpose of localizing a kidney stone and of ensuring the complete removal of multiple stones after lithotomy.

Quinby[1] (1925) was the first to point out the advantages of the film over fluoroscopy and actually demonstrate its practicability at the operating table. His method was simple. A small film (10 × 12.5 cm.) was wrapped in a sterile towel or slipped in a sterile rubber envelop and placed in the wound behind the kidney. Such a film bends readily, is difficult to insert in the wound, and lacks the rigidity necessary for proper manipulation of the kidney.

Benjamin[2] (1931) has recently described a cassette designed by Jaches. This resembles an ordinary cassette, but it is constructed on a miniature scale (3.75 × 4.75 inches). It is hinged in the usual manner of a cassette, is kept closed by two clasps at the opposite margin, and contains two intensifying screens. This cassette has the advantage of rigidity and permits a shorter exposure time.

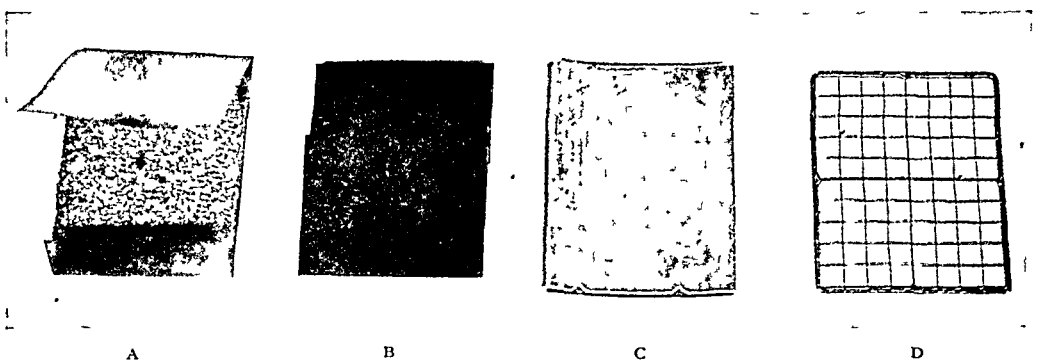


FIG. 1. Various parts of cassette and grating localizer (reduced 3½ times). A, Folded strip of film base. Within it is a thin layer of sponge rubber (portion of ordinary rubber bath sponge). Film base enables one to slip film pack into cassette without injury; sponge rubber effects necessary contact of fluorescent screen and film. B, Film pack, containing two films with a single fluorescent screen between them, protected from light by black paper. Only one of these films is affected by fluorescent light and two films of different roentgenographic densities result on exposure. Fluorescent screen is ruled off in centimeter squares with india ink. C, Slightly curved aluminum cassette. This is essentially an aluminum pocket without hinges or clasps. D, Centimeter grating, use of which is described elsewhere.

It appeared to us that the cassette method was capable of much greater development. The features desired in this cassette were (a) thinness, compactness, and rigidity to permit easy insertion in the wound; (b) shape to conform roughly with curvature of kidney; (c) elimination of troublesome hinges and clasps; (d) exclusion of light without the usual means of

[1] Quinby, W. C. A note on the localization of renal calculi by the aid of x-ray films made during operation. *J. Urol.*, 13: 59, 1925.

[2] Benjamin, E. W. Notes on the technique of x-ray control in the operating room. *J. Urol.*, 25: 165, 1931.

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light-proofing of the cassette by felt; (e) foolproofness, rendering it impossible to have the cassette inverted at time of

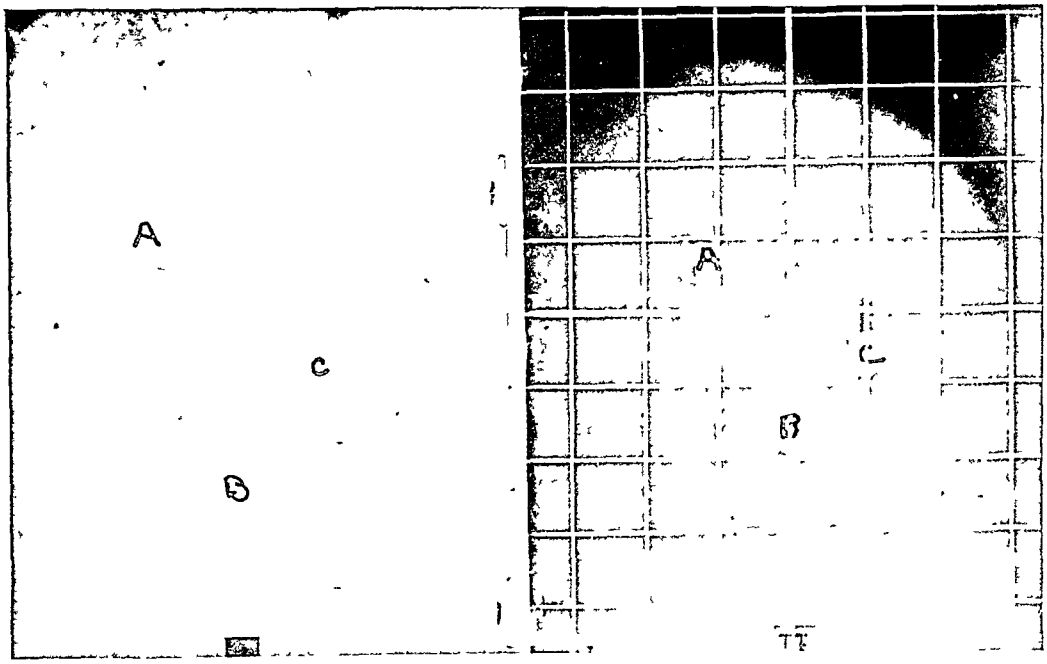


Fig. 2. Localization of improvised stone imbedded in model. Two local kidney films obtained from single x-ray exposure show two surface markers (A and B) and stone (C). Second film localizes all three in respect to centimeter pattern of fluorescent screen. After these films were obtained metal centimeter grating localizer was placed over wax kidney in such a position that two surface markers bore same relationships to centimeter localizer that is noted on centimeter patterns of film. Position of calculus was then determined, centimeter localizer being used as a guide. This position was marked on surface of kidney, localizer removed, and on drilling vertically downward with a small drill at this point, foreign body or calculus was recovered without difficulty.

exposure or to lose orientation of film after development, i.e., to confuse upper with lower poles of kidney, etc.; (f) insurance of proper exposure (all using this method are familiar with the ease with which a relatively non-opaque calculus may be missed on a dark, heavily exposed film and even a very dense stone on a weak, underexposed film. Time does not permit the retaking of poor films at operation and satisfactory films must be obtained at the first exposure); (g) more accurate localization of stones (in the past it has been disconcerting to the operating surgeon to have the radiologist demonstrate a stone on a film which the most painstaking probing or dissection of a kidney failed to reveal. Much kidney function has been sacrificed, at times the kidney has been removed, and the shock of the operation greatly increased as a result of the mutilation of a kidney containing such a stone or stones. All of these stones can be removed if located, and the new method should permit a more precise localization toward this end); (h) easy identification of artifacts and similar shadows (often local kidney films show confusing shadows which may simulate stones and at times the skill of the radiologist may be taxed to the utmost in establishing their identity); (i) simpleness of construction in attaining these ends.

A cassette to meet these requirements has been constructed in the following manner: A rectangular piece of aluminum 0.5 mm. thick, 10.8 cm. wide, and approximately 18 cm. long is bent to the form shown in Figure 1. Its sides are parallel and when folded in this fashion it makes a curved cassette measuring 10.8 cm. in length, 8.5 cm. in width, and 7 mm. in thickness. At the overlapping seam the

two pieces of aluminum are held by two rivets and the seam filled in with "plastic wood." One of the open ends is next filled in with this adhesive wood and when hardened the cassette is ready for use. This forms a slightly curved pocket with an open end, the inside dimensions of which are approximately 10.5 × 8.0 × 0.5 cm. Through the open end is placed a film pack which is composed of two films and one screen wrapped in black paper, the screen being placed between the two films in order to obtain a light and a heavily exposed film. To provide good contact for the screen a thin layer of sponge rubber is cut off the surface of an ordinary rubber bath sponge. The film pack and compression rubber are placed within the folded piece of film base in order to permit it to slip without difficulty into the pocket. This film base which has been cut the same width of the cassette is about 2 cm. longer than twice the length of the cassette. This leaves one end to project 2 cm. out of the cassette which aids in extracting the pack from the pocket after the exposure. During the exposure this free end is folded back into the pocket to be out of the way.

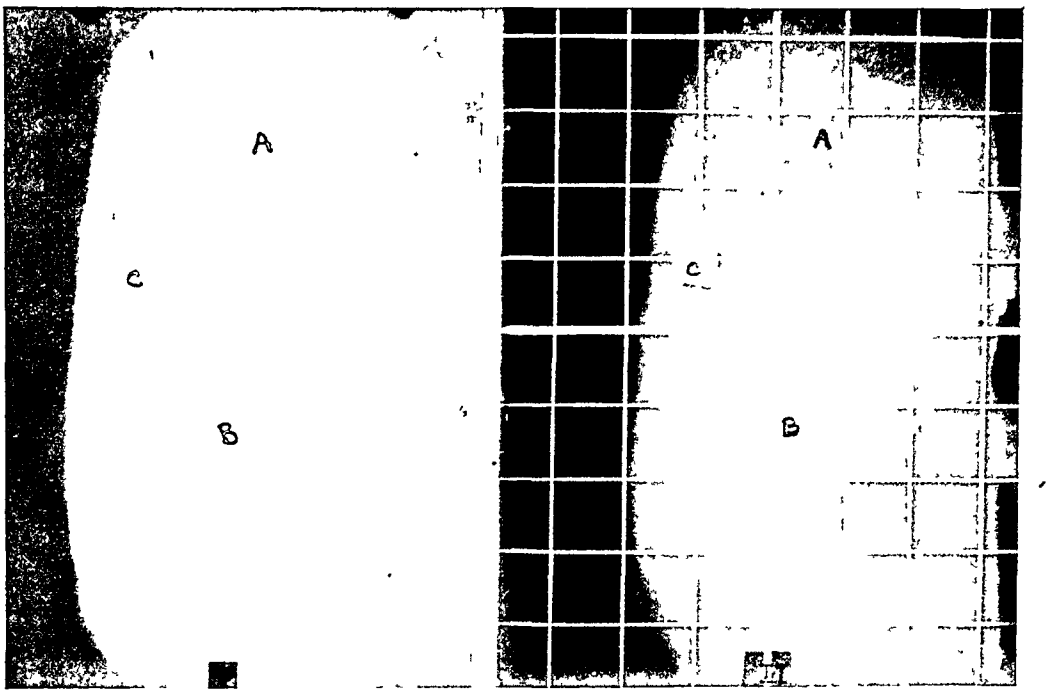


Fig. 3. Localization of improvised stone placed in human kidney obtained at autopsy. Markers (A and B) were placed on surface of kidney after inserting opaque body (C). Films show relationships of these markers to centimeter pattern of fluorescent screen. On placing centimeter grating localizer over kidney so as to maintain these same relationships, location of "calculus" beneath kidney surface is readily determined. This point was marked by placing a needle a short distance in parenchyma of kidney and under fluoroscope by directing needle vertically downward, point of needle was found to come in direct contact with "calculus."

Two notches cut in the concave (front or tube) side of the cassette at the open end will cast shadows on the film and aid in orienting the film after development, i.e. in preventing confusion of the upper with the lower end of film.

The description of the cassette is now complete except for the screen. The screen which has been cut down to proper film size (8.0 × 10.5 cm.) is ruled off with india ink into centimeter or half-inch squares. The middle lines in both planes are made slightly heavier than the others to aid orientation. Since this grating is ruled off on the sensitive side of the fluorescent screen, this same pattern will be noted on the film adjacent to it.

A metal grating of dimensions similar to those of the fluorescent screen is next prepared of suitable wire (Fig. 1). The square pattern of this grating is identical with that of the screen. This grating localizer can be sterilized readily and forms part of the sterile equipment of the operating table. The manner in which it is used at operation will be given later.

The cassette is slipped into a sterile rubber envelop with the open end of the cassette corresponding with the open end of the envelop. If the open end of the envelop was placed during the exposure toward the upper pole of the kidney, the film after development will be held with the two notches uppermost for viewing by the surgeon. The rubber envelop is about 7 cm. longer than the cassette. This end is folded back on itself once and is then rolled and held by any suitable clamp or the hand of the operator.

The *x*-ray beam passes through the following successive layers during exposure: concave aluminum plate, layer of film base, black paper, first film (direct exposure film not affected by screen), fluorescent screen with sensitive side away from tube side of cassette, second film (to be intensified by screen), black paper, compression rubber, layer of film base, convex plate of aluminum cassette.

The procedure at operation is simple. Two metallic markers are placed on the exposed side of the kidney about 3 or 4 cm. apart. They may be placed in any convenient position. The loaded cassette (closed end first) is placed in the sterile rubber bag by means of forceps, avoiding contamination of the outside of the bag. The end is folded and rolled as described. The cassette is slipped beneath the kidney. The *x*-ray tube is directly centered over the kidney avoiding the use of the periphery of the beam, and a film is taken at 26 inches (our operating limit for distance). The concave side of the cassette is toward the *x*-ray tube.

This single exposure yields two films, a lightly exposed film which is excellent for the identification of small or relatively non-opaque stones and a darker, more heavily exposed film which will show the centimeter grating of the fluorescent screen in addition to two opaque identification markers and one or more calculi.

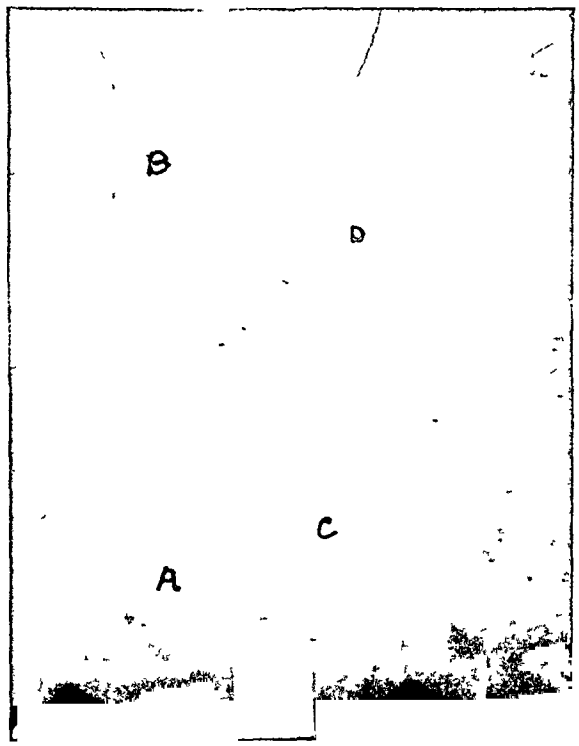


Fig. 4. Test film to determine extent that distortion plays in preventing correct localization. Wax kidney was placed on cassette and two holes (C and D) drilled vertically downward through kidney. Round lead markers were placed over openings on under side of kidney and similar square markers over openings on upper side. A film of such a kidney should show the two sets of markers superimposed if distortion is absent or minimal. Actual result obtained is shown in this illustration. It will be noted that these two sets of markers superimpose over most of their areas. This would indicate that regardless of distance which calculus may be from film, correct localization can be established. Maximal distortion obtainable with wax kidney of average size was determined by placing a brass rule with perforations exactly ½ inch apart longitudinally and transversely across top of kidney. When distance between perforations is measured on film, increase (due to distortion) is only 1/64 inch. This amount in operative practice is negligible.

The calculus is now localized on the film without difficulty by establishing its relations to the centimeter grating of the film. While holding the film for the operator for

this purpose, the film should be held with the correct end up, using the notches as the guide for orientation. The sterile, metallic grating is now oriented over the surface of the exposed kidney, the identification markers being used as a guide. The point on the kidney surface beneath which the calculus lies is now readily determined from the kidney film. A needle or other marker may be left to indicate this location before removing the metallic grating.

The accuracy of this method can be readily checked. A kidney modelled of paraffin was prepared and calculi or opaque material placed at random within the improvised kidney. After placing the two markers on the surface, films were obtained which localized these shadows. The paraffin kidney was then bored at those points indicated by the grating localizer, and these bodies recovered without difficulty. The results are illustrated in Figure 2.

Similar work was done with a human kidney obtained post mortem. The "calculus" was readily identified and oriented in regard to the centimeter pattern of the fluorescent screen (Fig. 3). When the grating localizer was correctly placed over the surface of the kidney, the "calculus" was located accurately and recovered.

It became apparent that the amount of distortion produced by the divergent *x*-ray beam was not sufficient to vitiate results at the distance used (26 cm.). It is important, however, to center the beam over the film, the periphery of the beam being avoided, and to place the cassette at a right angle to the axis of the *x*-ray beam.

To determine the amount of distortion produced when these requirements are met, the following experiment was carried out with the improvised wax kidney: The kidney was placed in the same position as for exposure of a film, and two holes were drilled vertically downward (perpendicular to the cassette) through the entire thickness of the kidney. Two small, square pieces of lead foil were placed over the uppermost termination of the drill holes, and two small, round pieces of foil over the lower end of these same holes. The shadows of these round and square markers should exactly superimpose on a film if distortion is absent. Films (Fig. 4) show the actual result obtained. The shadows of the square markers (farthest from film) are thrown very slightly away from the center, but the amount is so slight that the two sets of shadows superimpose over most of their area. This would appear to be sufficient.

After a more prolonged clinical use of this cassette, a report of actual operative results is contemplated. It may not be amiss to add a word of caution concerning extensive manipulation and probing of the kidney during the development of the film. Such manipulation should be avoided because of the danger of dislodging the stone to a new position. This method of locating stones should be particularly valuable in those cases where (a) the stone has become walled off from the tip of the calyx by connective tissue, and therefore cannot be reached by simple probing methods through the kidney pelvis, (b) stones imbedded in the parenchyma of the kidney, (c) all cases regardless of location of stones in which simple operative procedures fail to locate the stone, (d) all cases of multiple stones in determining their complete removal.

SUMMARY AND CONCLUSIONS

A simple, local, kidney cassette[3] has been described, which is not complicated in construction and can be made in any small laboratory at a trifling cost. Its construction does away with hinges, clasps and special light proofing. It assures proper exposure through the use of two films, one of which is affected by a fluorescent screen. Artifacts produced by films or screen are readily identified because a second film is always obtained and can be used as a control. Using the grating pattern present on one of the two developed films as a guide, the stone can be localized with greater accuracy.

[3] This cassette was designed at the suggestion of Dr. W. W. Scott for use on the Urological Service at the Strong Memorial Hospital. We desire to express our thanks to the Department of Pathology, particularly to Dr. W. B. Hawkins, for supplying us with fresh autopsy material for this work.

NEW INSTRUMENTS

METROPOLITAN HOSPITAL SPONGE-HOLDER*

F. M. AL-AKL, M.D.

NEW YORK CITY

THE object of devising this form of sponge-forceps is the obviation of certain difficulties encountered when using ordinary sponge-holders in preparing the skin of the abdominal wall for operation.

In preparing the abdomen, be it with iodine, picric acid or any of the other antiseptic solutions, the germicidal fluid is first allowed to trickle into the umbilical depression, then after the operative field has been painted, the umbilicus is mopped and the sponge discarded. To have the solution drip into the umbilical fossa one of two things is usually done. The thoroughly impregnated sponge is either carried instantly from the iodine glass and held over the umbilicus, allowing the excess of fluid to trickle into the umbilical depression, or the solution is squeezed out by crushing the sponge between the surgeons' fingers. Both forms of technique are very inadequate. In the former procedure the solution is sprinkled over the hand of the nurse holding the glass, the operating room floor, the surgeon's shoes or gown and the operating table sheets; furthermore the amount of solution falling may not be enough to guarantee a thorough disinfection of the many rugae and fossae of the umbilicus. In the latter procedure, the gloved hand is soiled which, handling the peritoneum later, may irritate the serous membrane.

The forceps in question holds the sponge gently but firmly by means of the six prongs. The impregnated sponge, once over the umbilicus, is crushed. The solution flows as freely as is desired by the compressing action of the two broad plates. The field is painted, then with the sponge in the umbilical fossa, the compression is released and the excess of solution reabsorbed by the decompressed sponge. Thus the sheets and floor of the operating room remain spotless, the umbilicus is thoroughly disinfected, the surgeon's gloves remain clean and non-irritating and the entire procedure is neater.

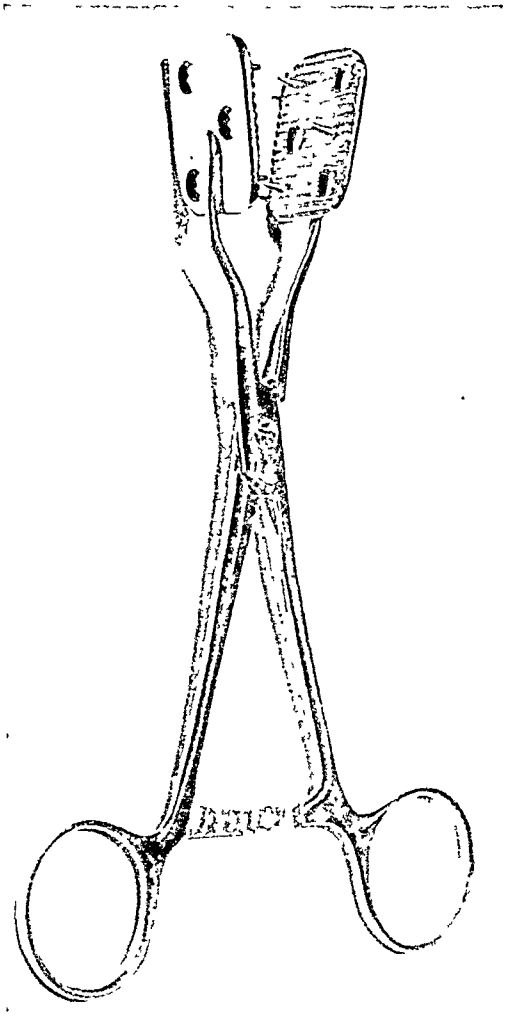


FIG. 1.

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EDITORIALS

PATHOLOGICAL CONSTITUTIONS

THEIR IMPORTANCE FOR SURGERY

TWO factors are usually considered in progress of surgical lesions and indications or contraindications for surgical procedures: (1) local conditions, such as enlargement of the involved organ, pain, tenderness, functional disturbances; (2) general conditions, viz.: age, respiration, temperature, blood count, blood pressure, urine examination, etc. and also the presence of concomitant conditions in remote organs, such as diabetes, tuberculosis, arteriosclerosis, hyperthyroidism, etc. The fact is frequently overlooked that the prognosis depends to a great extent also upon constitutional factors. For purpose of illustration let us take two apparently identical cases of appendicitis where the age of the patient, clinical symptoms, results of laboratory examinations and also operative findings resemble each other; one patient makes an uneventful recovery; the second one dies and the autopsy reveals status thymicus.

It is evident that constitutional pathology is of great importance in the practice of surgery as it may offer warning signs, which if disregarded may lead to a catastrophe. Consideration of individuality of patients is of importance for evaluation of the mode of action of surgical interference. Recognition of constitutional pathology is desirable not only because

it may help to establish a contraindication for an operation but also because some constitutional disturbances may be influenced therapeutically and thus better surgical risks may be created. It is true that constitution is predetermined by germinative cells but somatic fate of the individual may to a certain extent be influenced by extrinsic factors during fetal or postnatal life, e.g. interference with placental circulation, adaptation to climate, exercise, food, etc.

For obvious reasons hypersthenic, athletic types interest the surgeon less than hyposthenic or asthenic conditions caused by hypoplasia or qualitative inferiority of various tissues and organs. Hypoplasia comprises the following conditions: habitus asthenicus, enteroptoticus, neuroticus, phthisicus, thymicolymphathicus, hemasthenicus, etc.

Status thymicolymphaticus is the most important one from the surgical viewpoint. Some authors subdivide it into status thymicus and status lymphaticus, the latter being more frequent, but for our purposes this classification is superfluous. Recognition of such constitutional pathology at autopsy is easier than in vivo but certain signs are suggestive of a status lymphaticus. The head is rather small. The posture is relaxed, muscles poorly developed, chest flat and narrow, ribs have a steep direction, the epigastric angle is sharp, the abdomen flabby. Elasticity of skin is diminished. Lymphatic glands can be palpated on the neck, in the axillae, inguinal regions and other places; the lymph follicles on the base of the tongue are markedly enlarged; this is a very characteristic sign. Hypoplasia of genital organs, asthenic flat foot, varicose veins, varicocele, hemorrhoids, and formation of ganglia in the carpal region are frequently found. The blood pressure is usually low. Blood examination reveals the presence of lymphocytosis. Lability of the nervous system is usually found and psychoneurotic stigmata can be easily detected. Disturbances of the vegetative nervous system in such hypoplastic types manifest themselves in the form of dermographism, profuse perspiration, dilatation of pupils after adrenalin, etc. Roentgenological examination reveals a small heart, enteroptosis, abnormally long presence of epiphyses; persistent or hyperplastic thymus may also be found. During operation, the so-called Bartel's additional symptoms of hypoplasia may be found; état mamelonné of the stomach, long appendix, tortuous tubes. In the history of the patient frequent recurrences of tonsillitis or repeated infections of nasal sinuses or middle ear may be found: not infrequently there is an inclination to skin diseases.

Recognition of such types is important for two reasons: (1) Persistent thymus may be responsible for pathetic death after such trivial surgical procedures as tonsillectomy, circumcision, intravenous injections of salvarsan or other drugs. (2) Such types show an inclination to hyperplasia of connective tissue; adhesions form easily after laparotomies or operations on joints such as arthroplasty. Keloids may form in scars, causing unpleasant disfigurement after thyroidectomies; formation of keloids is especially embarrassing if an operation has been performed for cosmetic reasons. Such fibroplastic potencies are responsible for quantitative overproduction which compensates for poor quality of connective tissue or lack of restitution. Polyserositis observed after abdominal operations on hypoplastic individuals may be caused by inferiority of the lining cells of the serosa, increased production of fibrin or excessive formation of connective tissue in the subepithelial layer. In such cases French authors speak of fibroplastic or fibrous diathesis.

It may be said *cum grano salis* that the hypoplastic constitution offers certain advantages; increased inclination to formation of connective tissue results in facilitated encapsulation after local infections; hence perforations of the appendix, gall bladder or stomach lead to rapid formation

of adhesions preventing the spreading of infection. So-called covered perforations result; furthermore, the hyperplastic lymphatic apparatus serves as a mechanical filter interfering with dissemination of pathogenic microorganisms.

If stigmata of lymphatic constitution are found, a search for persistent or hyperplastic thymus gland is justified. Permanent or intermittent dyspnea may be caused by an enlarged thymus gland; the dyspnea usually has the form of inspiratory stridor. Sometimes percussion may detect the presence of an enlarged thymus. In doubtful cases a radiogram should be made.

If a persistent or enlarged thymus gland is found, an irradiation of this region preliminary to operation is advisable. If the surgical procedure can be postponed, an attempt should be made to raise the resistance of the body by outdoor life, gymnastic exercises and regulation of the diet. If an operation for cosmetic reasons in a hypoplastic individual is contemplated or an operation is planned for other reasons but the cosmetic result is important, as is the case in thyroidectomy, a small incision should be made on the thigh or some other covered place two weeks before the operation; thus an inclination to keloid formation may be detected. Although a keloid may form in one place and not in another, the aspect of the scar may serve as an indicator of fibroplastic tendencies of the organism. Inspection of vaccination scars or scars after previous operations is also of importance for evaluation of fibrous diathesis. As to the prophylactic treatment, hormones, especially extracts of the thyroid gland, have been applied hypodermically and also locally to the wounds. Other authors recommend foreign body therapy. Such prophylactic measures are of highly problematic value. Various methods of dealing with postoperative adhesions have been suggested, e.g. applications of pepsin with or without hydrochloric acid; amniotic fluid; olive oil, camphor oil, etc. Apparently no efficient method is available for prevention or treatment of intraperitoneal adhesions as the authors of all the methods suggested overlooked the fact that in the majority of cases the tendency to form adhesions is due to constitutional and not to local factors.

For hemasthenic types abnormally thin vessels and deficient contractility of the walls of the vessels is characteristic; such factors probably play a role in hemophilia in addition to a disturbance of the chemical composition of the blood. The constitutional inferiority of hemasthenic individuals involves the entire vascular apparatus and probably also the reticuloendothelial system. X-ray irradiation of the spleen before operation in such persons is followed by beneficial results.

A complete physical examination of patients before operations should include examination of lymph follicles of the tongue; lymphocytosis should also attract attention. Positive findings suggest a further search for stigmata of lymphatic constitution. If such are found, a radiogram of the region of the thymus gland should be made. If a persistent or hyperplastic thymus is present, it should be irradiated before any surgical procedure. Thymic hyperplasia with or without objective symptoms is potentially dangerous to life. If patients with status thymicolymphaticus are operated on, special care must be taken to avoid traumatism; tissues should be handled gently in order to avoid the formation of adhesions. This applies chiefly to laparotomies and operations on joints.

Joseph K. Narat

CORRESPONDENCE

OXYGEN LACK IN HYDRAULIC VICIOUS CIRCLE

To The Editor:

In The American Journal of Surgery, June, 1932, in "Hydraulic Vicious Circle as it Develops in Acute Appendicitis," I describe the deoxygenation which is present in hydraulic vicious circle and how it causes infection.

The obstructed appendix fills with fluid. Hydraulic pressure arrests the circulation. Arrested circulation means edema, effusion, more fluid in the appendix, more pressure, more effusion, hydraulic vicious circle.

Oxygen is quickly dissipated from an arrested blood stream. Leucocytes and tissue cells need oxygen for their very life. Practically all the pus-producing bacteria normally present in the appendix grow anaerobically and a perfect anaerobic culture tube is thus provided for them. They promptly multiply; the asphyxiated tissues succumb and an acute infection results, being favored by the dilating influence of the distention and by the increasing stasis of the blood stream.

The correllary of these mechanics is: There will be no infection so long as normally functioning capillaries furnish an adequate supply of oxygen.

I referred to the oxygen determinations of Burget and am pleased to add at this time those of Visscher. I hope some one will make some determinations in acute appendicitis and in abscess.

Dr. Maurice B. Visscher, in charge of the Department of Physiology in the College of Medicine, University of Illinois, Chicago, writes to me under date June 23, 1932, as follows:

My Dear Doctor: I want to report some observations which I have made on the oxygen tension in a loop of intestine in the anesthetized dog. Ringer's solution was introduced into a loop of gut under a pressure of 100 mm. of Hg. Originally a Ringer's solution was equilibrated with oxygen at 140 mm. of Hg. tension. In one-half hour in dog number 1 the oxygen tension had fallen to 60 mm. Hg. The following table gives the results in four dogs:

OXYGEN TENSION IN INTESTINAL LOOP (MM. HG)

	Beginning	*Half Hour*	*One Hour*
No. 1.....	140	60	
No. 2.....	140	28	12
No. 3.....	140	28	20
No. 4.....	140	15	4

Several other experiments yielded identical results. I think it is safe to say that the oxygen in the fluid in the loop of gut under pressure, so great as to stop the flow of blood through the loop, diminishes to practically zero in an hour after the pressure is established.

I feel quite sure that these results will be of interest to you, because I believe they confirm very directly your views as to the rapidity with which a tissue deprived of its blood supply uses up all of its oxygen.

I believe that my description in the above-named article is the first mention of this factor in infection. At least I can find no previous record of it, and my discussion with several well-known bacteriologists confirms that opinion.

C. Van Zwalenburg.



JOSEPH LOVELL

JOSEPH LOVELL was born in Boston, December 22, 1788. He graduated from Harvard in 1807, studied medicine under Dr. Ingalls of Boston, and graduated from Harvard Medical School in 1811.

The year after his graduation Lovell entered the Army, as surgeon of the Ninth Infantry. During the greater part of the War of 1812 he was in charge of the General Hospital at Burlington, Vermont, where, in 1814, he became Hospital Surgeon.

Following the War of 1812 the medical department of the Army was formally organized, and in 1818 Lovell was appointed Surgeon-General. He lost no time in making sweeping changes and organizing his department. He revised and reissued the regulations for its government. In 1821 he further improved and elaborated the organization. The form of organization which Lovell finally perfected continued in force until 1861. In 1834 he inaugurated a system of examinations for admission to the medical corps, which system of examination has continued to the present time.

In Packard's "History of Medicine in the United States"[1] we read: "Alcoholism was rife and a cause of much sickness. Surgeon-General Lovell, who was a strong advocate of temperance and whose labors and reports eventually resulted in the abolition of the rum ration, possibly unconsciously exaggerated the harm resulting from drink, but he ascribed more than half of the deaths in the Army during the decade 1819–1828, to 'this war against nature.'"

Lovell administered the affairs of the medical department in the early part of the Seminole War.

Among other worthy works an outstanding one was Lovell's encouragement of and assistance to Dr. William Beaumont, whose work on gastric digestion resulting from his observations upon Alexis St. Martin is one of the classical sagas of American medicine.

We read of Lovell that "In all his relations, whether as Christian philanthropist, profound scholar, skillful surgeon, experienced officer or true hearted gentleman, he was one of whom the Medical Staff may always be proud."

Joseph Lovell died October 17, 1836.

T. S. W.

[1] N. Y., Hoeber, 1931.

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JOSEPH LOVELL

[1788-1836]

The American Journal of Surgery
N S Vol XVIII, October, 1932



[From Fernelius' Universa Medicina, Geneva, 1679.]

BOOKSHELF BROWSING

THE ROLE OF EXPERIMENTS ON ANIMALS IN THE TREATMENT OF DISEASE*

MODERN SURGERY

A. STEPHENS GRAHAM, M.D.

ROCHESTER, MINNESOTA

INTRODUCTION

THE surgeon of today is capable of operating, with a relatively high degree of success and a remarkably low mortality rate on almost all parts of the human body. Operations include those on the brain, spine, stomach, liver, gallbladder, appendix, pancreas, spleen, kidneys, arteries, veins, and nerves. Furthermore, such operations, with but few exceptions, owe their origin, as well as their success, to animal experimentation.

In fact, modern progressive surgery, as developed from animal experimentation, represents strikingly a marked deviation from the deeply worn and uncertain paths of empiricism. Empiricism as used here implies a principle of treatment devoid of experimentation and based on an application of logic to facts born of observation, in instances in which the observations are too often superficial and the logic unsound. "The art of observation," says Dumas in a letter to Pasteur, "and that of experimentation are very distinct. In the first case, the fact may either proceed from logical reasoning or be mere good fortune; it is sufficient to have some penetration and the sense of truth in order to profit by it. But the art of experimentation leads from the first to the last link of the chain, without hesitation and without a blank, making successive use of Reason, which suggests an alternative, and of Experience, which decides on it, until starting from a faint glimmer, the full blaze of light is reached."

At about the middle of the nineteenth century anesthesia made its timely appearance as the first vital factor in the development of modern surgery. Anticipated and sought for since the beginning of surgery, it was but natural that the use of anesthetics should at once become almost universal. Operations that had been impossible could now be performed; for the most part these operations were in the abdomen and were first performed and perfected on animals. The technique of some of these operations approached perfection, and even today is used with little or no change, and yet the procedures were soon discarded, because of the havoc wrought by infection in the abdomen and in the wounds incident to the opera-

* This is the sixth of a series of articles on the value of animal experimentation in medical progress. The next article will appear in an early issue. This paper was prepared for publication in a lay journal.

tion. The mortality following these early operations was distressingly high, and the pain that had been so mercifully eliminated from the operation by the anesthetic, soon manifested itself in the pus-laden, heavily infected wounds. Yet the surgeons of this early period still spoke of "laudable" pus, and labored in ignorance of the antiseptic method of surgery.

This state, however, did not exist long. Barely a quarter of a century later, Pasteur's researches on fermentation and his epoch-making animal experiments with bacteria were applied by the surgeon Lister in the building of an antiseptic method of surgical procedure. The "full blaze of light" is not yet reached; but through constant experimentation and the application of its results to repair of the human body, the glow of perfection becomes brighter.

Since the very earliest times the surgeon has been first the anatomist, second the operator and third the pathologist. He has dissected human bodies when possible; but in the early centuries this was frequently difficult because of the superstition and ignorance which clogged the wheels of progress. Where such conditions prevailed the surgeon resorted to animal dissection in order to glean knowledge applicable to human beings. As a pathologist he became interested in the investigation of the dead, in the hope that by determining the effects of disease some method of cure might be discovered. Gradually pathology grew into a great independent science, which exerted and still exerts a powerful and stimulating influence on surgery. The animal experiments of the great German pathologist Virchow (1821–1902) play no small part in this rapid growth.

The surgeon became interested in bacteriology when the antiseptic method was introduced into surgery, and he has maintained that interest ever since although he leaves its progress and its interpretation to the skilled bacteriologist. In more recent years the important influence of physiology has appeared, to remove the mantle of secrecy from nearly every organ in the body and to demonstrate the various functions by animal experimentation. Operations today must be more than merely technically perfect; they must meet all the exacting requirements of physiology. Nor can the average surgeon in a consideration of the broad scope of this fundamental science hope to master all of its intricate details; he must be guided by those who make this their life work. Thus has surgery emerged from the baleful darkness of ignorance and uncertain empiricism, into a scientific art of sure beneficence, closely associated with, and ever influenced by, these basic sciences.

It is fitting to state here that modern surgical experiments on animals are conducted with the same careful consideration that is shown by the surgeon in the care of his patients. Complete surgical anesthesia is invariably maintained to forestall the possibility of pain and discomfort; and conditions of strict antisepsis are adhered to in all operations. Particular attention is paid to the care of the animals after operation; their quarters are hygienic and comfortable, and their diets are carefully adjusted to individual needs. In fact, in order that the experimental observations may be entirely applicable to the treatment of diseases of man, they must be made under conditions closely analogous to those of the human patient.

SURGERY OF THE ABDOMEN

The abdomen was appropriately named from the Latin verb meaning to hide. For a period of more than a thousand years, from the beginning of surgery to about 1875, this important cavity of the body has managed well to withhold its secrets from the surgeon. The manner in which this veil of secrecy was finally lifted through the combined agencies of anesthesia and antisepsis has been related. It might well be asked, however, if surgeons in the early days could operate on blood vessels in

order to "bleed" a patient, and make holes in the skull, trephine, to relieve increased pressure within the head, and amputate limbs, all without anesthetics, why he could not likewise operate on the abdomen and its contents. The answer is simple: Any one of the foregoing operations could be consummated with extreme rapidity, often within a few minutes, whereas abdominal operations required considerably more time and more exactness. The pain incident to such a long procedure would be almost unbearable and conducive to fatal shock. Furthermore, the violent movements of the stomach and intestine in the absence of an anesthetic would alone make the operation practically impossible.

The rôle of animal experimentation in the actual development of abdominal surgery is a story of brilliant ingeniousness and of infinite humaneness. Every basic principle essential to operations in the abdomen, and almost every subsequent new procedure, was first carefully tested on animals. Such procedures were so entirely new as to have barely a single precedent, even as related to fundamental principles, by which the surgeon could be guided. There was practically no parallel between the meager surgical procedures on the skull, the blood vessels, and the extremities, and those he was about to carry out on the various abdominal organs. There were many pertinent questions of a basic nature that needed immediate answer such as: Could an opening be made into the stomach or the intestine, or a piece of either be removed, and the edges then be brought together and sewed, and if so what material would best serve the purpose for sewing; silk, linen, cotton, or "catgut"? Could the entire stomach or a large portion of the intestine be removed? These and many other questions were promptly answered by numerous carefully conducted experiments on animals, and the fact that the investigators were for the most part among the leading surgeons of the day did much to hasten the universal acceptance and practical application of their observations on animals.

The year 1875 marks the beginning of new surgery of the digestive tract, the stomach and the intestines. Experiments on animals performed in that year and the succeeding year had far-reaching effects on surgery as applied to human beings; they initiated a vigorous continuous wave of progress. In the first year Tschertneisky-Barischewsky cut out pieces of intestine in thirty-five dogs; twenty-nine recovered, a most inspiring accomplishment. In the next year Gussenbauer and Winiwarter removed portions of the stomach in a series of dogs, and examined the stomachs of those that lived several months later. These experiments are of historic significance and of fundamental importance. They demonstrated that the raw edges of the cut stomach were not attacked by the digestive juices of this organ, that certain materials were best suited for stitching the cut edges together, that contraction or distortion of the stomach did not occur at the site of the operative scar, and that the pylorus could be removed, along with a considerable portion of the stomach proper, without any ill effects on the processes of digestion. The last observation was destined to play a vital part in the strenuous campaign that was soon to be inaugurated against cancer of the stomach. But in the meanwhile considerable interest was manifested in the simpler operations on the intestines.

Hueter, in 1876, operated on and sewed up the intestine in man, although without success. He was soon followed by Czerny and the great surgeon Billroth, both successful in removing portions of the intestines and reuniting their severed ends. The results of these experiments and subsequent first trials on man quickly spread to all parts of the world. Surgeons in every country performed such operations and promptly reported their failures and their successes. Other experiments were immediately begun with a view to bettering the technic of joining the severed

portions of the digestive tract in order to prevent fatal leakage. The experiments were carried on in this country chiefly by Senn, Abbe, Brockaw, Ashton and Murphy. Senn, besides improving the technic of uniting the ends of the bowel which had been cut, contributed valuable information concerning the rapidly fatal condition of intestinal obstruction. He produced experimental obstructions in the bowel of animals comparable to those in man, and demonstrated how these could be removed and in case of cancer or gangrene the manner in which the diseased bowel itself could be best removed.

In this early period cancer of the stomach, more than any other one disease, occupied the surgeon's careful consideration. The toll from this disease was appalling and the manner in which the patient died was pitiful. Usually there were months of suffering accompanied by gradual starvation. The cancer usually commenced near the outlet of the stomach, and slowly encircled it so that by degrees the pylorus became obstructed until eventually no food could pass out of the stomach. Pean in 1879, following the experimental procedure of Winiwarter, removed the pylorus for the first time in man; the cancer was far advanced and the patient died shortly afterward. Billroth a year later performed the third operation, the first successful one, which necessitated removal of about a third of the stomach, and then reconnected it to the intestine. Since then, there have been hundreds of such operations successfully performed, not alone for the removal of cancer, but in recent years for the removal of ulcers of the stomach and the duodenum. Since many patients, however, fail to reach the surgeon before the disease has already spread throughout the body, surgery in these cases can be no more than palliative.

Sometimes the cancer is in the esophagus or in the cardiac end of the stomach, and in either case it prevents nourishment from entering the digestive tract. As early as 1837 an opening through the abdomen into the stomach was suggested as a means of feeding these patients. The operation of gastrostomy was first performed by Sédillot in 1849 and again by him four years later; unfortunately both operations terminated fatally. He was severely criticised by the profession and the public for his boldness; he had not first tested and perfected the operation on animals. During the next quarter of a century twenty-seven similar operations were attempted with twenty-seven failures. It is true that the lack of an antiseptic method of performing operations was in part responsible for this high mortality, but most important was the lack of a fundamental knowledge of surgery. The mortality remained high, even after the introduction of antisepsis, until 1890, when the first thoroughly scientific operation of this nature was perfected on animals. From then on there has been a constant lessening of the mortality to the present comparatively low rate.

The method of feeding directly into the stomach is not alone useful in cases of cancer; it has proved a life-saving measure in those pathetic instances, too often seen today, of young children who have swallowed lye or some other corrosive, carelessly left within their reach. The corrosive usually results in stricture of the esophagus that prevents solid food and often even liquid food, from entering the stomach. Immediate establishment of a gastrostomy or enterostomy is demanded and until the stricture has been gradually stretched, often requiring months, all nourishment is supplied through a tube directly into the stomach or intestine.

Another milestone in the splendid progress of abdominal surgery is the advent of modern surgery of the kidney. The manner in which this was brought about represents one of the finest examples of the direct benefits to mankind derived from animal experimentation. Simon, in 1869, was confronted with the proposition of removing an apparently healthy kidney from a healthy woman. Many years before, the

tube leading from one of the kidneys to the bladder was injured, with the result that the ureter had grown into the abdominal wall and urine escaped continuously over her person, a most deplorable condition. Whether man could survive with but one kidney was unknown. There were cases in which one kidney had become gradually destroyed by disease, but in such instances the other kidney slowly became larger and eventually was capable of assuming the work of both. To remove a healthy kidney suddenly was a different matter. It was not even known how the kidney could best be removed since it is situated beneath the intestines and close to the spine. From the results of experiments on a number of dogs, Simon decided that one kidney would suffice and experiments on the cadaver showed the best mode of entrance to be through the loin. He then perfected on dogs the operation for removing the kidney. At last, thoroughly satisfied that he was justified in operating, he removed the kidney that was giving rise to the abdominal discharge and the patient lived in health for eight years. These few experiments opened wide the way to entirely new surgical procedures.

Most of the advances in abdominal surgery so far reported have followed the original investigations on animals by Germans and Austrians; as a result they led the world in surgical progress for years. It is therefore gratifiying to relate the inspiring and humane studies of Parkes and Senn, both Americans who were first to demonstrate by animal experiments the advisability, in fact the duty, of the surgeon to explore the abdomen in cases of gunshot and stab wounds. During the Civil War all such wounds were left essentially to the mercy of Nature, for surgical intervention was considered unjustifiable. In many body wounds and in many diseases Nature plays an important rôle and the surgeon relied on this unseen aid; but in wounds of the stomach and the small intestines she saved barely an average of one patient out of each hundred. Of the sixty-four soldiers who were reported to have sustained wounds of the stomach during the Civil War, only one lived. Of more than 650 who sustained wounds of the intestines only five of those whose wounds were in the small intestine survived, and only fifty-nine of those whose wounds were in the large intestine, a mortality of 90 per cent! Parkes, in 1884, with this in mind, conducted a series of systematic and carefully performed experiments on animals in which the animal was deeply etherized, then a small shot was projected through the abdominal wall, the abdomen opened, and the wounds of the intestines, blood vessels, and other abdominal organs treated by appropriate surgical methods. The results were reassuring and instantaneous in their influence on many surgeons in the treatment of similar cases. The next year Bull successfully repaired seven wounds in the intestines of a man shot with a pistol. Senn carried the experimental study still further and developed a method in animals whereby the perforated bowel could be definitely diagnosed before operation. Since this time there have been hundreds of such wounds successfully treated and recovery has been reported after as many as twenty-two gunshot perforations of the bowel.

It is interesting to compare the respective mortality rates incident to wounds of the abdomen during the Civil War and of those during the World War. In the former there were no abdominal operations recorded and the death rate was more than 90 per cent. In the latter there was a large number of such operations and the mortality was 43.32 per cent. Yet "intra-abdominal wounds in the war just ended," reads the official report of the Medical Department of the World War, "were definitely more grave than in preceding wars because of the nature of the projectiles used." Machine-gun bullets and high-explosive shell fragments in particular were responsible for the frightful wounds inflicted within the abdomen. Colonel Gibson, U.S.A., stated that in nineteen

months of active service overseas he did not come in contact with an incident in which recovery followed an intra-abdominal wound unless operation was resorted to.

Many other abdominal operations have originated through animal experimentation but lack of space will not permit their discussion here. In recent years experimenters have had in mind chiefly the improvement of abdominal operations already in use. The basic principles of operations which originated in the latter part of the nineteenth century still obtain in most instances, but the procedures have been so carefully perfected on animals that the mortality even in the hands of the average operator is remarkably low. Then, too, many surgeons are now trained by first operating on animals, in order that their skill may be developed to a high degree before operations are attempted on man. Even the skilled operator often resorts to animal tests before undertaking an operation that is new to him and which is technically difficult. It is significant that the newly organized National Board of Medical Examiners* requires the applicant for licensure to operate on animals, when these are available, in the presence of an examining board, that it may be determined whether the applicant is thoroughly acquainted with the basic principles underlying abdominal surgery. Requirements such as these tend greatly to raise the surgical standard of perfection and thus further to safeguard the patient whose life is placed in the hands of the surgeon. Indeed, these methods of animal experimentation, and practice in surgical technic, are not only of the utmost practical significance, but what is more, they are vitally humane.

* This Board was organized with the purpose of establishing a standard qualifying examination of such a high character that the certificate awarded successful candidates could be safely accepted by all the State Boards of Medical Licensure as an adequate qualification for the practice of medicine.

SURGERY OF THE CHEST

The surgery of this great cavity, enclosed by the ribs, advanced slowly and cautiously, far behind the brilliant progress in abdominal surgery. This was in part due to the greater complexity of the chest in comparison with the abdomen, and in part to the lack of definite knowledge concerning the physiology of respiration. The biologic aphorism, "no life without breathing," indicates in a general way the importance of respiration. Man may survive for weeks without food and for days without water, but can exist only a few minutes if deprived of air. Naturally the surgeon would meditate long before dealing surgically with a process so vital as respiration. But this was not the sole reason for concern. The heart and its great vessels, so intimately related anatomically to the lungs and the bronchi constituted a grave potential danger in any operation on the chest. Operation on the heart itself was considered no more than a fantastic dream.

Although remedial surgical procedures in the region of the chest were slow in developing, there was urgent need for them. Foremost was the need of removing foreign bodies, either swallowed and lodged in the esophagus, or drawn down the air passages into lungs. Usually these accidents occurred in children and constituted a pitiful group of sufferers. Safety pins, buttons, marbles, pieces of toys and, in fact, almost every small object one can imagine has managed to find its way into the lungs. The ingeniously devised instruments, the bronchoscope and the esophagoscope, which today usually make operation unnecessary in such cases, were not available until comparatively recent years. The perfection of these remarkable procedures depended to a considerable extent on animal experimentation. Then, there were abscesses of the lung, either spontaneous or following pneumonia. Death often claimed the miserable victim. Spontaneous rupture into the bronchi, Nature's method

of ridding the body of the accumulated poison, was the best hope, but Nature was not always benevolently inclined.

Cancer of the esophagus was also badly in need of some means of treatment. Buried behind the lungs, bronchi, and important nerves, and closely attached to the great blood vessels, the esophagus was extremely difficult of access. There was, also, need for an efficient method for repairing wounds of the chest, wounds from all varieties of accidents, from gunshots and from stabs.

Sauerbruch, in 1904, supplied the vigorous impetus so essential to the splendid new surgery of the chest that was destined to make its appearance in the first quarter of the present century. This was in the form of a specially constructed air-tight chamber in which operations could be performed on the chest without hindrance to the processes of breathing; the air in the chamber could be either exhausted or compressed in order to prevent collapse of the lung. The perfection of this unique and highly scientific apparatus was made possible through the availability of the dog as an experimental animal. Willy Meyer modified this chamber and within its confines removed part or all of one lung in twenty-six dogs, with twenty-two recoveries (84 per cent). These tests further inspired the surgeon, lending courage to those who only awaited such a confirmation of Sauerbruch's original and excellent method. Almost immediately there were reports of remarkable operations performed in these chambers, operations by noted surgeons, such as von Eiselsberg and Trendelenburg. Both of these men first developed methods on animals and then applied them to man. But here it is necessary to record the objection to the apparatus. It was available to only a chosen few since it was bulky, stationary and expensive, and consequently it was installed in only a few of the larger universities in this country and on the continent. Still, the knowledge derived from physiologic experiments on animals in the Sauerbruch chamber was of inestimable practical benefit to the surgeon in subsequent years. It was in this manner that he became acquainted with the normal action of the heart and lungs, and their reaction to disease and treatment.

Five years after Sauerbruch's memorable contribution, Meltzer and Auer, of the Rockefeller Institute in experimenting on dogs, provided the much needed simple procedure which could be substituted for the chamber in operations on the chest. It provided for artificial "ventilation of the lungs," produced by the introduction of a small tube into the trachea which would permit a warmed mixture of oxygen and ether to be forced gently through into the lungs. Collapse of the lungs during an operation was thus forestalled and the normal processes of respiration continued without impediment. This was indeed a long stride forward. Immediately, experiments on animals by thoroughly competent surgeons multiplied. Operations were devised for the removal of cancer of the esophagus, for injuries and diseases of the great blood vessels and the heart, for cases of advanced consumption and for other serious conditions. Carrel, of the Rockefeller Institute, who by his remarkable ingenuity and dexterity, has contributed so much to our knowledge of surgery of the blood vessels and the transplantation of organs, was one of the first to utilize the Meltzer-Auer method in operating on the chest of animals. In 1910 he reported a series of experiments in which the thoracic aorta was severed completely and the continuity immediately reëstablished by sewing the cut ends together. Many of these animals survived. This study pointed the way to repair of the large blood vessels of the chest, and suggested other possible operations which time and more experience will undoubtedly prove of value. These methods of repair were used to great advantage in the World War and subsequently in civil practice.

The foregoing relates to fundamental discoveries made on animals and the ready

application of these discoveries to the alleviation of human suffering. The investigators were pioneers, workers in an intricate and little understood field of surgical endeavor. A world calamity was necessary in order to put many of their experiments to a thorough test, to elaborate on them through observations on the maimed, and through further study of animals. The recent official report on surgery of the chest during the World War to the Surgeon General of the United States Army is in reality a vivid description of recent experimental procedures in the cavity of the chest. In France, under the leadership of Brigadier General J. M. T. Finney, a laboratory of Surgical Research was established where many of America's most illustrious scientists, experimental physiologists, pathologists, chemists, experts on anesthesia, and surgeons, concentrated their efforts in the investigation, through animal experiments, of devices and new methods to meet the ever increasing demands. There was no end to problems. In each battle new implements of death were brought into action: the various types of poisonous gas, liquid fire and shrapnel, and always the devastating machine gun fire.

Wounds of the chest were numerous and it was essential that a simple yet highly efficient method be invented for keeping the patient asleep during operation and at the same time preventing the lungs from collapsing. Ether had its disadvantages; so had chloroform; some other anesthetic was needed. Captain Gwathmey started experimenting with nitrous oxide gas. In reading the official report relating to these experiments we find the following interesting evaluation: "Animal experimentation showed clearly that administering anesthesia under tension, particularly when the chest is opened, was dangerous if the gas or ether was given in increased concentration" . . . and "So closely related is the physiology of man and dog,* in spite of the gross anatomical differences in the pleura and mediastinum, that the principles established experimentally were directly applicable to the wounded."* The American Red Cross Nitrous Oxide Apparatus thus perfected by Captain Gwathmey, entirely through animal experimentation, and adopted by the Army, fulfilled every requirement and proved to be of inestimable value to the surgeon operating on the chest.

Now that the anesthesia problem was solved, all efforts were centered on standardizing methods for repairing the thousands of chests mutilated by the instruments of war, which were ever increasing in numbers and in power of destruction. How could the lung best be sewed up after the removal of an injured portion? There was little precedent to follow and then there was lacking a unanimity of opinion as to the best methods. However, thanks to the excellent animal experiments of Willy Meyer, and others, in which measures were devised for removing a portion or all of one lung, a foundation was laid on which further to build our knowledge. But never before was the surgeon confronted with such a variety of severe injuries of the lung as were brought to him at the battle front; lungs rent with bullets, macerated by fragments of exploded shells, and not infrequently, gangrenous in portions due to injury and subsequent delay in treatment. Should he remove the entire lung in these cases of injury? That probably would have often been the line of least resistance; but was there not a way to save most of this vital organ; who knew but what the other lung might some day become diseased? The modern trend of surgery is to conserve every piece of injured tissue that is not definitely proved irreparable. It was said of that venerable master of surgery, the late Dr. Halsted, who wielded such an important influence in the establishment of this conservative trend, that "he stood firm on the ground that the nature of the material upon which the surgeon exercised his handicraft is too noble and too precious to be insulted with impunity or to allow even

the smallest living particle to be wasted." By animal experimentation and clinical application he was ever demonstrating this.

The same problem had confronted earlier surgeons in the face of severe injuries to the arm and leg. Twenty years ago many limbs were amputated which today would be saved in their entirety, or greater part. The problem was solved then as it was solved by these workers in France, relative to injuries of the lung, by animal experimentation. So striking were these earlier experiments that it seems best to digress in order briefly to relate them. Abbe, before the New York Academy of Medicine in 1894 almost amputated a dog's foreleg, leaving the limb connected with the body by nothing but the artery and vein. He wired the bones together, sewed muscles and nerves end-to-end, and placed the leg in a cast of plaster. After four months this almost amputated limb was perfectly united. A few years later, Carrel and Guthrie, of the Rockefeller Institute, went even further. They removed the entire leg of one dog and transplanted the corresponding leg of a second dog to the body of the first one. Here, besides rejoining bone, muscle, and nerve, they had added difficulty of rejoining blood vessels. Yet, the transplanted limb grew firmly to its new stump.

Such experiments as these inspire the surgeon with confidence; they justify an application of the principles involved to operations on human beings. Although limbs have not been transplanted in man, many severely maimed arms and legs have been saved to serve an economic usefulness. The same is true with regard to injuries of the lungs. In the surgical research laboratories, behind the battle lines in France, studies were made of injured lungs in animals to determine to what extent these wounds may be conservatively repaired by simple sutures, without gross amputation of lung tissue.

In time these and other serious difficulties were mastered. Many injured lungs were conservatively treated, as healing was found to be kindly where there was no infection; ingeniously conceived methods for stitching the frail substance of the lung were invented; repair of the injured wall of the chest was studied; experiments were performed to determine whether the surgeon was justified in draining the chest of large accumulations of blood, as would often follow the passage of a single bullet through the chest, yet without inflicting serious injury to its contents. These constitute but a few of the most important problems these splendid investigators set out to solve, but they suffice to show the great value of animal experimentation in the progress of such procedures during the War. It is worthy of note that with these workers the gruesome realities of war dulled not a whit those fine instincts of kindliness, gentleness, and compassion toward the animals that they were compelled to substitute for the maimed soldier, in experimenting. These animals, most of whom were condemned to destruction in the municipal guillotines, were treated with the same consideration as were accorded the injured soldier in the base hospitals. "The dogs were invariably operated on," reads the official report, "in ether or nitrous oxide narcosis, and were protected against unavoidable distress by a free use of morphine before and after operation."

We leave France and the battle field for at home a great scourge, influenza, was ravaging the country; it killed more than did the implements of war. It spread to the remotest village and attacked man, woman and child. In its wake were left thousands maimed in a most miserable fashion by the dread empyema. The army cantonments held large numbers of such patients and in the early days a few survived; but there were still more who died. In March 1918, the Surgeon General of the Army sent questionnaires to each cantonment in order to determine their various methods of treatment of empyema and their respective death rates. The average mortality was 30.2 per cent;

in one locality it was as high as 90 per cent.

What was to be done? These soldiers were dying by the thousands. Were they to be experimented on? Apparently not. But then, what? The Empyema Commission, created by the Government to solve the problem, deliberated; conflicting views as to treatment were presented; the treatment at that time consisted of making an opening in the diseased chest as promptly as the condition was diagnosed, to permit free drainage of the pus. But too many soldiers were dying. Immediately, action was demanded.

The Commission detailed two of its members, Graham and Bell, to solve this urgent problem through animal experimentation. They at once set about to the task of producing experimental empyema and pneumonia in dogs. In one series of cases ten dogs with empyema were operated on; another series of ten was not operated on. From the results of these experiments it was clearly shown that to open the chest early in the face of pneumonia was unwise. Other experiments demonstrated the size of the opening in the wall of the chest that might be compatible with life. Still other experiments covered certain additional controversial points in treatment.

The treatment suggested by these observations on animals was in full accord with the deductions that the Commission had previously made from a study of the accumulated data relative to empyema in the soldier. But the Commission did not feel justified in putting into full action, as mandatory for all the cantonments, the methods of treatment born of these deductions. We now know that the death rate in cases treated in harmony with the results of the experiments of Graham and Bell was reduced to the extremely low rate of 3.4 per cent. Yet before this, an average of thirty soldiers died out of each 100 afflicted with empyema. Is it possible that any one could wish that these experiments had not been performed in order that the animals thus sacrificed on the alter of humane progress in surgery could now live?

It might be well in passing to mention that during the last ten years there have been many other investigators studying the mechanism of the chest and its contents. Some of these have corroborated the observations of Graham and Bell, while others feel that they cannot accept in their entirety their physiologic interpretations. But this does not imply doubt with regard to the inestimable value of their experiments in causing the marked reduction in the death rate in empyema. Snyder, working at the Institute of Experimental Medicine of The Mayo Foundation, performed experiments which led him to maintain a somewhat different viewpoint as to the mechanism of the cavity of the chest. He stated, however: "The opportune work of Graham and Bell was undoubtedly responsible for the saving of many lives during the recent epidemic of influenza."

SURGERY OF THE HEART

It is fitting that discussion of the heart should come at the close of the discussion of thoracic surgery; for it is only within recent years that surgery of the heart has received serious consideration. Although still in its early infancy, recent experimental studies betoken a splendid future for this type of surgery. Allen, of St. Louis, has within the last few years developed a method on dogs whereby the heart can be entered with an ingeniously devised instrument that permits the operator to view the entire inside of the heart during the operation and yet does not allow a drop of blood to be lost.

Heretofore the surgeon was required to operate with extreme swiftness, taking not longer than one or two minutes, and all manipulations inside the heart were of necessity done by the sense of touch alone. There have been comparatively few operations performed on the heart; for there have been few surgeons so bold as to

adventure on this uncharted sea of great potential dangers. But there has been a definite need for this type of operation. There is a large group of sufferers who are condemned to long years of invalidism, or to early death. Such patients have diseased heart valves which become thickened and distorted by disease, or partially destroyed, the valves leak and eventually the heart reaches the limit of its ability to compensate for an ever increasing disability.

In the light of the remarkable reconstructions consummated by the surgeon in nearly every part of the body, it is but natural to expect that the time is not far distant when his skill will be applied toward lengthening the span of life in that large group of persons whose heart valves are diseased. These valves have been operated on in the dog and there will be many repetitions of such operations; eventually all doubt will be dispelled and every weak point in the theory of the procedure will be covered with sound experimental reassurance; then man will profit from these long years of cautious testing and retesting.

BOOK REVIEWS

RECONSTRUCTION OF THE BILIARY TRACT. By Edmund Horgan, M.D. N. Y., Macmillan & Co., 1932.

This is a comprehensive, well-written, and well-illustrated monograph on an important topic. There are few situations more tragic than that of the man or woman who comes with a common duct so scarred that it will no longer carry bile. If something is not done these people must go downhill and die a lingering death. Many procedures have been devised which will return these patients to health, and they are all well described by Dr. Horgan. The reviewer's only regret is that there are so few surgeons skillful and experienced enough to do this type of work; he wishes there were more men in the country interested enough to buy this book and repay the devotion of the writer and the publisher.

ATLAS DER RÖNTGENSTEREOSKOPIE. (Atlas of Roentgenstereoscopy.) By Max Cohn, Physician in Charge of the Roentgen Dept., State Hosp. in Friedrickshain-Berlin. First Series of 20 negative film transparencies, with a folding stereoscope. Leipzig, Georg Thieme, 1932.

This is a laudable attempt to bring to the medical public education in what roentgenology can accomplish toward clearing up obscure diagnoses. The twenty films chosen for the first series have been happily selected to exemplify the value of the roentgen diagnosis, and especially of the stereoscopic method as applied to this diagnostic work. Aside from cinematography, there is nothing in the way of photographic teaching methods to compare with the use of stereograms. It will be interesting to see what the future brings forth in this venture. Among the interesting cases presented are included ectopia of the kidney due to retroperitoneal lipoma; perforation of a tuberculous kidney; demonstration of the duodenum by the method of Pribram; echinococcus of the lung; fracture-dislocation of the lumbar spine, with myelography to demonstrate compression of the spinal canal at the site of the fracture; cysticerci of the muscles of the thigh and pelvis.

DIATETIK BEI CHIRURGISCHEN ERKRANKUNGEN. (Diet in Surgical Disease.) By F. W. Lapp and H. Neuffer. Berlin, Julius Springer, 1932.

There is no question about the desirability of better dietetic care of patients who have submitted to operation. Just at present, with most of the dietitians worried sick over vitamins, about all the average patient, recovering from a gastric or intestinal operation, can get in an American hospital is coarse spinach, salad, fruit, and cod liver oil. It is high time that we should return to our former habits of thinking of the digestibility of a diet, at least often enough so that we will see to it that the material put into a handicapped digestive tract is not full of woody material.

There are many good suggestions in this book and it should be of interest to every hospital dietitian. There are many diet lists and many recipes. What bothers the reviewer is the lack of good sense on the part of men who seem to think they can fit one diet to a patient with an anal fissure and another to a patient with hemorrhoids. This assumption of omniscence is a common mistake made by writers on dietetics. An author seems to think that in order to sell his book he must include a diet list for every known disease. Actually when no three "authorities" on diet will agree as to what should be given to a patient with any one disease, what is the use of trying to devise one diet for gastroptosis and another for enteroptosis? The man with good sense must see that the science of dietetics has not advanced to the point where any such accuracy is possible. He who thinks he can do this sort of thing should go shooting with three sizes of shotgun; one for ducks, one for grouse, and another for pheasants.

GROSSE ARZTE: EINE GESCHICHTE DER HEILKUNDE IN LEBENSBILDERN. By Prof. Henry E. Sigerist. München, J. F. Lehmanns, 1932.

This work by the new Professor of Medical History at Johns Hopkins, is, as its title indicates, a history of medicine in biographic

form. Short biographies are given of forty-seven important figures in medicine from Imhotep through Pettenkofer. The author has the remarkable faculty of saying much in few words and making it interesting at the same time. His viewpoints are stimulating and for every German-reading physician and student this book is to be recommended for supplementary reading, no matter how large or how meager his knowledge of the history of medicine. Students of the subject will be impressed with the author's literary ability and tyros in medical history will learn much from this short volume which, it is hoped, will soon be translated into English. We look forward to many interesting contributions from Dr. Sigerist after he has become acclimated to America.

Lectures on Endocrinology. By Walter Timme, M.D. Ed. 2, N. Y., Paul B. Hoeber, Inc., 1932.

In the Preface to this work, the author states: "It is my desire that the book retain its present form and that the material be chosen for easy and rapid reading not only by the student for whom it was originally intended, but also by the busy medical man. Therefore only the most salient features of the advances in the subject of Endocrinology have been added, and are given as succinctly as may be."

Dr. Timme has succeeded well in his intention. In ten chapters, as follows: I. Introduction; II. The Thymus Gland; III. The Pineal Gland; IV. The Thyroid Gland; V. The Parathyroid Glands; VI. The Suprarenal Glands; VII. The Pituitary Gland; VIII. The Gonads; IX. Compensatory States; X. References, he gives an up-to-date, rational and consecutive concept of the present status of the science of Endocrinology.

The medical man will obtain in this volume just the insight he needs into this subject which leads so many authors into drawing on their imagination rather than adhering strictly to the facts. Dr. Timme has resisted all such temptation and has presented what the reviewer feels to be just the book that is needed by the average practitioner.

The Use of Lipiodol in Diagnosis and Treatment. By J. A. Sicard and J. Forestier. Lond., Oxford Univ. Press, 1932.

This book of 192 pages of text and 43 additional pages of bibliography and index is a result of many years' experience on the part of the originators of this method. The senior author died before completion of the work and Dr. Forestier has brought it strictly up-to-date. The subject is covered with unusual thoroughness. With the growing use of lipiodol, all roentgenologists will want this book at hand for reference and it is so written that it will also be found of interest and value by the general practitioner and the general surgeon. The authors present the case for lipiodol conservatively and modestly.

The American Illustrated Medical Dictionary. A Complete Dictionary of the Terms Used in Medicine, Surgery, Dentistry, Pharmacy, Chemistry, Nursing, Veterinary Science, Biology, Medical Biography, etc. With the Pronunciation, Derivation and Definition. By W. A. Newman Dorland, A.M., M.D., F.A.C.S. Ed. 16, Phil., W. B. Saunders Co., 1932.

An up-to-date dictionary is a requisite in the library of every physician, especially one who writes. The sixteenth edition of the famous "American Illustrated Medical Dictionary" has just appeared, completely revised and enlarged with the collaboration of Dr. E. C. L. Miller of the Medical College of Virginia. Hundreds of new words have been added and a more or less careful scanning has revealed few, if any, errors. The new edition of this dictionary is sure to retain its place as one of the most valuable reference books in the physician's library. The typography and format leave nothing to be desired.

For editors and authors, this book is indispensable whereas for the practicing physician it will answer many questions not answered in other volumes.

A Text-book of Anatomy and Physiology. By Jesse Feiring Williams, M.D. Ed. 4, Phila., W. B. Saunders Co., 1932.

The fourth edition of this work has been revised and brought up-to-date, particular attention having been paid to the chapter on Digestion, and Physiology has been elaborated throughout the entire work. The book

is well illustrated and simply written. It is a remarkable presentation of the subject for those for whom it is intended—premedical students and nurses. Each chapter is followed by practical exercises, questions and references for further study.

Having stood the test of four editions, this book may be safely recommended as a reliable guide to the elementary study of anatomy and physiology.

MINOR SURGERY. By Frederick Christopher, S.B., M.D., F.A.C.S. With a Foreword by Allen B. Kanavel, M.D., F.A.C.S. Ed. 2, Phila., W. B. Saunders Co., 1932.

The second edition of Christopher's "Minor Surgery" has been made, according to the author, "a more useful and up-to-date volume." The question of Minor Surgery is still a debatable one and many surgeons will resent some of the inclusions in this volume under this title. The information contained will be found up-to-date, reliable and accurate although, of course, the scientific surgeon will want to refer to other books for more detailed descriptions.

PHARMACOLOGY OF THE MEDICINAL AGENTS IN COMMON USE. By Stanley Coulter, PH.D., SC.D. Indianapolis, Lilly, 1932.

This pharmacology is prepared with special attention to the needs of the medical student. The main part of the text deals with individual drugs and is followed by an appendix of tables and miscellaneous information useful to the medical student. The subjects are arranged alphabetically for quick reference. Under each title there is a terse statement of the constituents of the drug, its physiological action, dosage, and brief mention of its more important therapeutic uses.

In no sense is this book intended to supplant the larger standard texts on pharmacology. On the other hand, it is the hope of its author and publishers that the use of the pocket-sized book will so interest the student in the subject that he will be led to closer studies of the great authorities on pharmacology.

THE CAUSATION OF CHRONIC GASTRO-DUODENAL ULCERS; A NEW THEORY. By J. Jacques Spira. N. Y., Oxford Univ. Press, 1931.

A reader who dips into this book here and there is likely to be impressed for a while by the many quotations from authors who have written well on the physiology and pathology of digestion. But when these quotations are studied more carefully it is found that they are poorly strung together, and sweeping conclusions are made from premises of very doubtful value. The author should have read a statement made years ago by either Huxley or Darwin to the effect that one cannot take statements made by various writers about different things and add them and subtract them with the same confidence that one uses in adding or subtracting angles in geometry or parts of equations in algebra. Even if all the statements were fairly correct they might not be able to stand the new strain put upon them, and actually, as in the present case, many of them are so wrong and so out-dated that a writer who is really conversant with the literature of his subject would never think of using them to bolster up his argument. Thus a man who knew anything about anthropology would never think of saying that, "In spite of a large consumption of fat, the Eskimo is small, stunted, and has a low resistance to disease, in contradistinction to the sun-baked but semi-starved Arab, who is straight limbed and displays a magnificent physique" (pp. 32–33).

A sample of the author's vague logic is to be found in Chapter 15 in which, on the basis of one family history, he thinks he had proved something or other. Just what it is, is doubtful. He says: "In any case, environment, i.e. the local condition, must be considered of primary importance, as it constitutes a constant influence to which the tissues have to adapt themselves in the process of subsistence and evolution, and one must agree that 'Nature shows signs of purposive adaptation to environment.' In the present case, where it is suggested that a local influence is at fault, it must be considered, if not as the controlling factor, at least as the factor that releases or sets in motion the action of other influences. But since it can be affirmed that indiscretions in the diet are undoubtedly the immediate cause of the majority of recurrences, it would be only logical to deduce

that some item in the diet is the initial factor in the causation of the disease."

The book is so puzzling in many places and the thesis of the author is so poorly presented that the reviewer is hazy about it even after two readings of the book: one for his personal information, and one for the purposes of this review. Apparently Sir Humphry Rolleston realized this defect and in his introduction tried to help the reader by summarizing the argument as follows: "Fat when introduced into the stomach regularly causes regurgitation of the duodenal contents, including bile, into the stomach; and bile salts, when mixed with the acid gastric contents, damage the mucous membrane of the stomach. Evidence in favour of the accuracy of these various events is provided by reference to the work of others, and attention is drawn to the analogous acute damage effected in the pancreas when bile passes into its ducts. The proximity of the duodenum, as compared with the stomach, to the biliary papilla is brought forward as an explanation of the greater frequency of duodenal ulcers, and of the greater difficulty in treating them as compared with gastric ulcers. Fat inhibits gastric movements, delays digestion, and though valuable from its contained vitamins is necessary in small quantities only. It is further insisted that as olive oil does not contain any vitamins there is not any rational basis for the modern treatment of gastric ulcer therewith, which was introduced into this country by Dr. Hurst in 1907. The familial incidence of peptic ulcer is explained by the author as due to identity of the food conditions (namely excess of fat) in the affected families."

Apparently Spira feels that fat is responsible for all forms of indigestion because on page 64 he says, "Careful and prolonged observations have strongly impressed on my mind the fact that all patients suffering from gastric disturbances are, without exception, unable to consume fat."

The most curious feature of the book is that here is a man who is claiming that he has found a new treatment for ulcer, a treatment that is better than anything that has gone before, and yet when one reads the six pages which make up Chapter 18 on "Clinical Experience" one finds practically nothing about the method used. A few cases are reported in which the bare statement is made that the treatment was begun and was carried out with excellent results. Apparently small doses of alkali were given, but this is nothing new. Finally he says, on page 69, "As far as the regime itself is concerned, it can be most easily worked out by anyone who understands the rationale of the theory, and there is no difficulty in teaching every patient to master the basic principles which will guide him in the choice of his food. If properly chosen this dietary will conform in all respects with what a regime should aim at as laid down by Fernandez Martinez, viz., (1) Non-irritating to the gastric mucosa; (2) rapid evacuation; (3) excite the minimum of secretion; (4) remove the hydrochloric digestion of mucous membrane; (5) fight the infection."

Surely this should be plain enough for anyone to follow and it doubtless represents a remarkable advance in our knowledge of the subject! Unfortunately for the advancement of his theory the author shows few signs of having any clinical acquaintance with the manifold problems of ulcer or of the extensive literature on the experimental production of the disease; furthermore he fails to face the troublesome fact that, thus far, fat seems to have been one of the most useful and helpful constituents of most of the diets that have been used in the treatment of ulcer.

Medical Electricity for Students. By A. R. I. Browne. Ed. 3, Lond., Humphrey Milford, Oxford Univ. Press, 1931.

This is the third edition of an elementary work on "Medical Electricity for Students." It is intended primarily for technicians and the book well serves its purposes although many of the instruments and much of the technique shown differ from American usage.

Recent Advances in Anaesthesia and Analgesia. By C. Langton Hewer, M.C., B.S. (Lond.). Phila., P. Blakiston's Son & Co., Inc., 1932.

As its title indicates, this is not a textbook of anesthesia but rather a review of the newer and most up-to-date methods. Taking for granted the knowledge on the part of his readers of the elementary facts in anesthesia, the author describes the new methods and

the physiological and pathological aspects. The book is up-to-date and yet thoroughly conservative. The keynote of his text is expressed by the author in his preface when he states: "'Safety first' as a slogan may be open to criticism for some purposes, but it is no bad motto for the anaesthetist." With this every surgeon will agree and will wish that the phase was imbedded in the mind of every anesthetist. Conservatively, yet thoroughly, the book discusses all the new drugs and methods. The bibliography at the end of each chapter is well selected. Not a bad volume to have at hand when the pros and cons of modern anesthesia are under discussion.

ANATOMY OF THE HUMAN ORBIT AND ACCESSORY ORGANS OF VISION. By S. Ernest Whitnall. Ed. 2, Lond., Oxford Univ. Press, 1932.

This revised edition of a book, which has been a favorite with ophthalmologists since 1921, has been completely brought up-to-date and many of the illustrations have been replaced by new and better ones. Well written and unusually well illustrated, with a thirty-three page bibliography and a good index, this book will be indispensable to those interested in the subject.

BOOKS RECEIVED

All books received by THE AMERICAN JOURNAL OF SURGERY are listed in this column as soon as possible after their receipt and this must be considered as adequate acknowledgement. Books that the Editor considers of special interest to our readers will be reviewed in a later issue.

ANATOMY OF THE HUMAN ORBIT. By S. Ernest Whitnall, M.D. London, Humphrey Milford, 1932.

ANNUAIRE GENERAL DE L'UNITERSITÉ LAVAL POUR L'ANNÉE ACADÉMIQUE 1932–1933. Quebec, L'Action Sociale, Lim., 1932.

ATLAS CHIRURGISCH-PATHOLOGISCHER RONTGENBILDER. By Prof. Dr. Rudolf Grashey. Munich, J. F. Lehmanns, 1932.

BIOGRAPHISCHES LEXIKON, vol. 4. Berlin, Urban & Schwarzenberg, 1932.

CARDIAC OUTPUT OF MAN IN HEALTH AND DISEASE. By Arthur Grollman, M.D. Springfield, Ill., Chas. G. Thomas, 1932.

CLASSIC DESCRIPTIONS OF DISEASE. By Ralph H. Major, M.D. Springfield, Ill., Chas. G. Thomas, 1932.

CURATIVE VALUE OF LIGHT. By Edgar Mayer, M.D. N. Y., D. Appleton & Co., 1932.

ESSENTIALS OF PATHOLOGY. By C. Russell Salsbury, M.D. N. Y., D. Appleton & Co., 1932.

HANDBUCH DER GESAMTEN UNFALLHEILKUNDE. Ed. 2. By Fritz Köing, M.D. and Georg Magnus, M.D. Stuttgart, Ferdinand Enke, 1932.

HANDBOOK OF THE VACCINE TREATMENT OF CHRONIC RHEUMATIC DISEASES. By H. Warren Crowe, M.D. London. Oxford Univ. Press, 1932.

INDIVIDUALITY OF THE BLOOD IN BIOLOGY. By Leone Lattes, M.D. (Trans. by L. W. Howard Bertie, M.D.) London, Oxford Univ. Press, 1932.

KOSMETISCHE OPERATIONEN. By Ernst Eitner, M.D. Vienna, Julius Springer, 1932.

LIVRE D'OR À L'OCCASION DU JUBILÉ DE VINGT-CINQ ANS D'ACTIVITE CHIRURGICALE DU DR. THEO L. PAPAYOANNOU. Naumburg-Salle, Germany, Lippert & Co., 1932.

MATERIA MEDICA PHARMACOLOGY, THERAPEUTICS AND PRESCRIPTION WRITING. Ed. 3. By Walter A. Bastedo, M.D. Phila., W. B. Saunders, 1932.

MENTAL DEFICIENCY DUE TO BIRTH INJURIES. By Edgar A. Doll, M.D., Winthrop M. Phelps, M.D. and Ruth Taylor Melcher, M.D. N. Y., Macmillan Co., 1932.

MESENTERIC LYMPH-ADENITIS—A CLINICAL STUDY. By J. P. Strömbeck, M.D. Stockholm, P. A. Norstedt & Sons, 1932.

PRINCIPLES OF ORTHOPEDIC SURGERY FOR NURSES. By James Warren Sever, M.D. N. Y., Macmillan Co., 1932.

TEXT-BOOK OF PATHOLOGY. By William Boyd, M.D. Phila., Lea & Febiger, 1932.

PERIPHERAL NERVE INJURIES

LEWIS J. POLLOCK, M.D., AND LOYAL DAVIS, M.D.

TENTH INSTALLMENT

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Chapter XXIX

THE BRACHIAL PLEXUS (*Continued*)

Sensory Symptoms

The sensory loss of brachial plexus lesions is rarely coextensive with the motor disability. It is not as reliable an index to the extent or severity of the injury as is motor loss.

Soon after injury the sensory loss is quite extensive but it rapidly shrinks so that some months later the motor loss may be complete for a certain localization whereas little sensory loss may be found (Figs. 247–251). At times complete sensory loss may be found and the motor disability be slight. This is particularly true of lesions of the inner cord. In the latter case, as has been found in ulnar nerve lesions, the sensory loss is often complete when but slight motor disability exists. Sensory loss coextensive with motor paralysis was usually seen in lesions of the fifth or the fifth and sixth cervical roots. The same condition was met with when the injury affected the fully formed peripheral nerves (Figs. 252, 253 and 254).

Attention should be called to the fact that with the exception of root lesions and primary cord lesions the sensory loss consequent to injuries of the brachial plexus corresponded neither to a root nor a peripheral distribution.

In upper plexus lesions, usually root or primary cord, the sensory loss corresponded closely to a root distribution. At times even when the biceps was not paralyzed there was an analgesia over part of the sixth cervical segment. In a lesion which produced a paralysis of the deltoid, biceps and brachioradialis muscles, the sensory loss never extended to that part of the hand usually attributed to the sixth cervical segment. On the other hand, when in addition to the biceps the pronator radii teres was paralyzed, the sensory loss was found over the radial side of the palm, including the index finger and thumb (Fig. 255). That this represented the sensory supply

of the outer head of the median nerve is further supported by such cases as showed, among other things, a paralysis of the muscles supplied by the median nerve, with the exception of

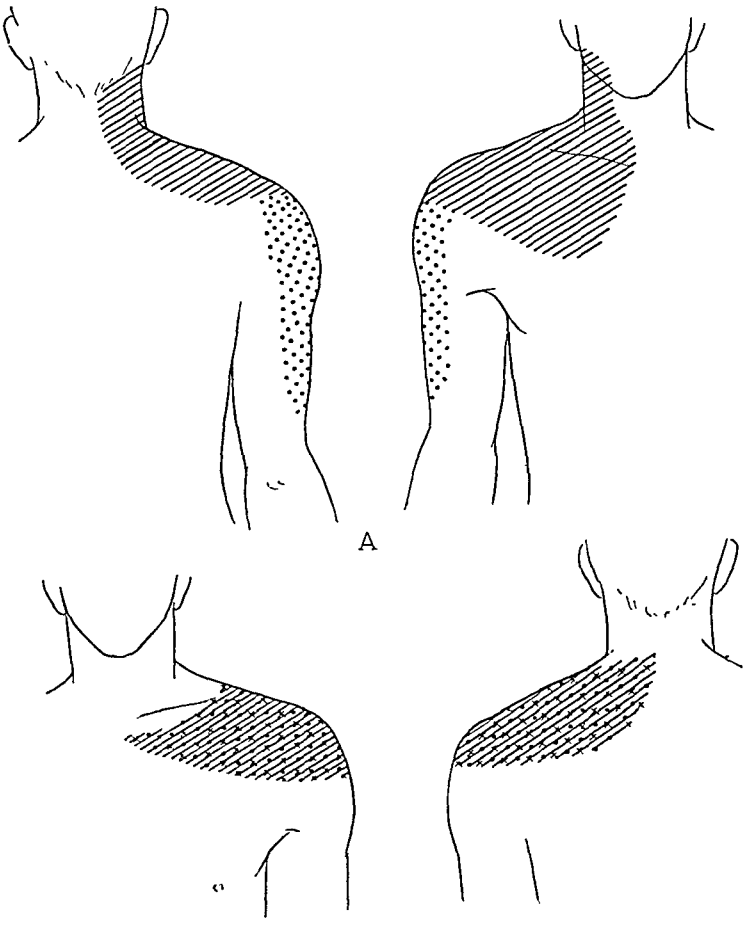


FIG. 247.

A. Sensory loss in lesion of cervical third and fourth roots.
B. Sensory loss in lesion of cervical fourth root.

the pronator radii teres alone or together with the flexor carpi radialis. In these instances sensation was unaffected over the radial part of the palm.

In Figures 256 and 257, the former a lesion of the inner primary cord and the latter a lesion of the posterior and inner cords, the radial side of the palm was sensitive as well

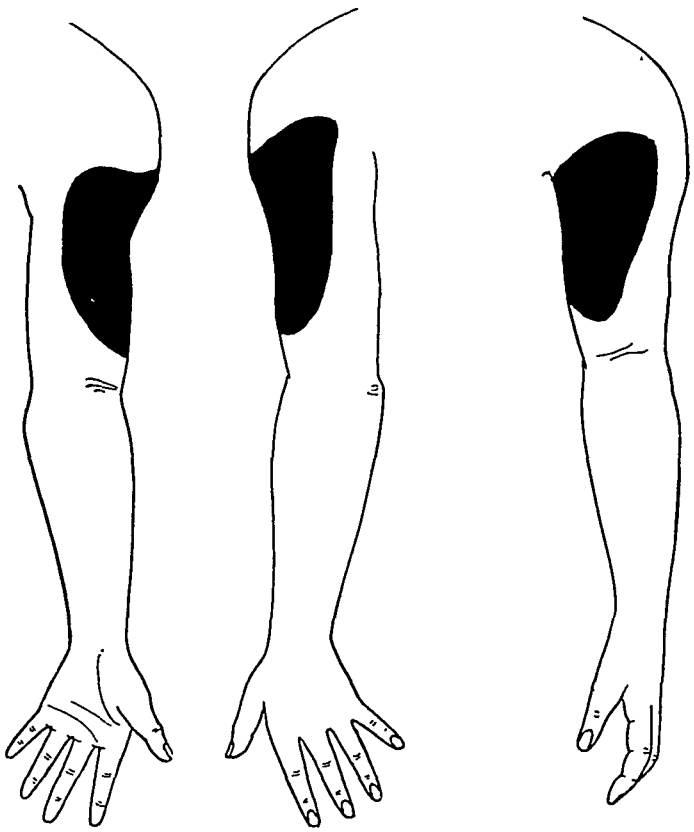


FIG. 247.

C. Sensory loss in lesion of cervical fifth root.

as the palmar surface of the thumb. It may be concluded, therefore, that the sensory area of the outer secondary cord of the brachial plexus is represented by the sensory distribution of the musculocutaneous nerve and that part of the median which supplies the radial surface of the palm, the thumb and part of the index finger. This part of the median sensory supply would then correspond to the anterior division of the seventh cervical root (Fig. 258).

The inner cord is represented by the sensory supply of the medial cutaneous nerves of the arm and forearm, the ulnar, and the median, with the exception of the thumb, part of the

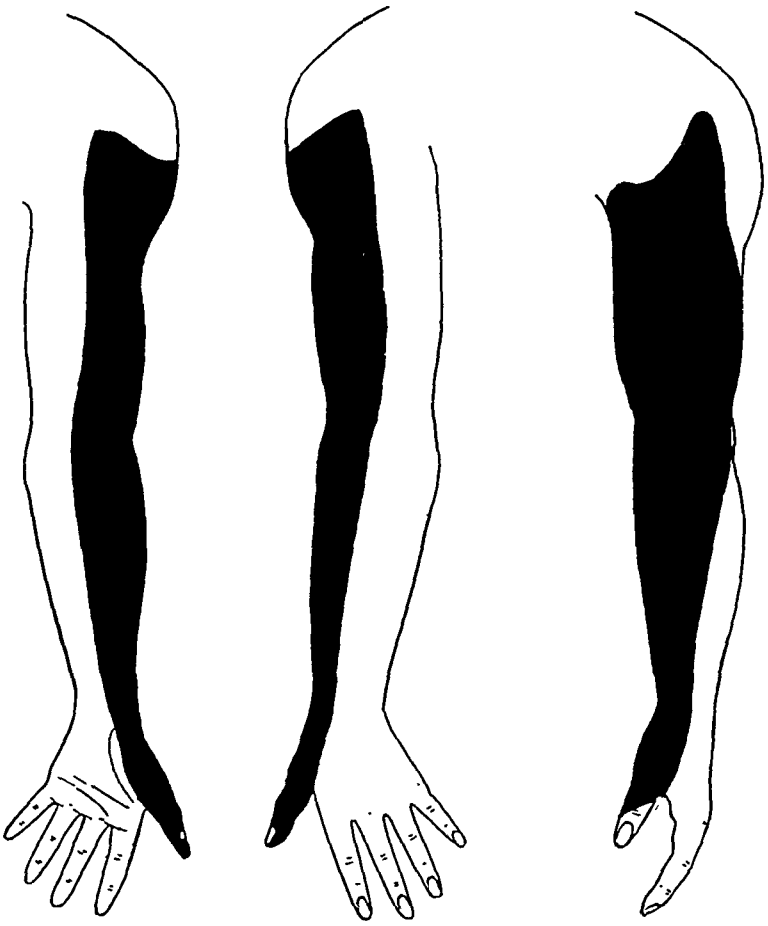


FIG. 248. Sensory loss in lesions of cervical fifth and sixth roots.

index finger and the radial side of the palm.

SIGNS OF RECOVERY

Lesions of the brachial plexus have a tendency to recover. Often the recovery is only partial, but it may become complete in from a few months to two years. In upper plexus lesions the biceps and brachialis anticus recover before the brachioradialis. The rhomboids, pectoralis major, latissimus dorsi and teres

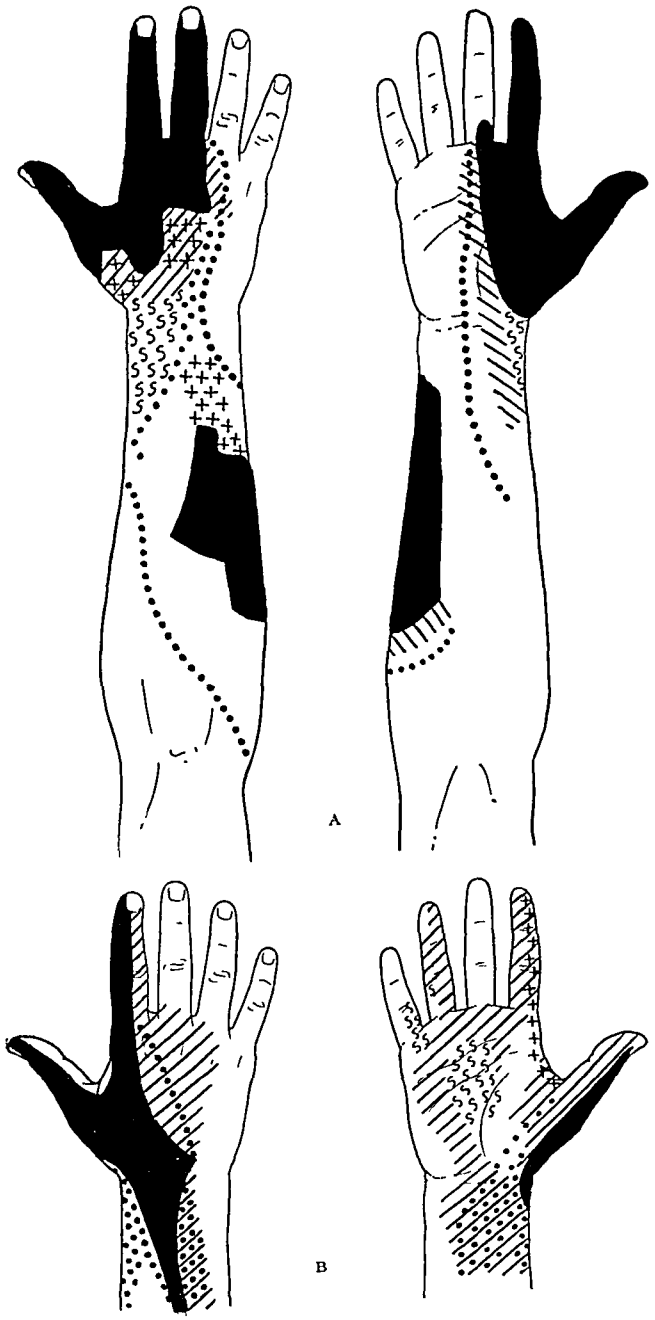


FIG. 249.

A. Sensory loss in lesion of cervical sixth and seventh roots.

B. Sensory loss in lesion of cervical sixth and seventh roots and partial lesion of cervical eighth root.

major are often only partly paralyzed and frequently recover early. The deltoid, infraspinati and brachioradialis require a longer time to recover completely.

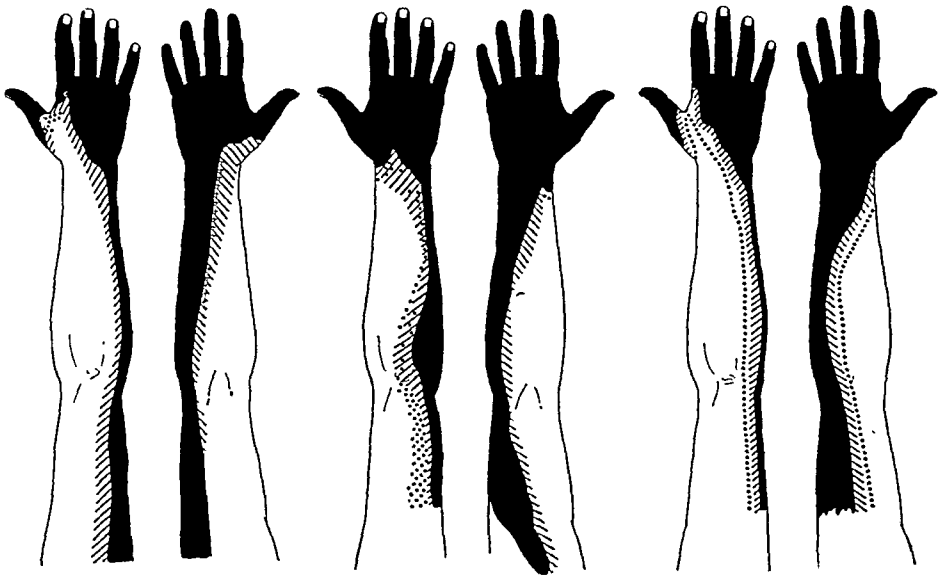


FIG. 250. Sensory loss in lesions of cervical seventh and eighth, and dorsal first and second roots.

The characteristics of the recovery of sensation in brachial plexus lesions are shown in Figure 259.

COMBINED LESIONS OF THE BRACHIAL PLEXUS AND SPINAL CORD

Brown-Séquard paralyses frequently occurred as the result of gunshot injuries. It is to be expected that injury to the roots may occur on the side of the injury to the spinal cord. At times, however, cases have been observed in which the symptoms, sensory and motor alike, corresponded neither to a root lesion, an intramedullary or a peripheral nerve lesion (Gordon Holmes). Such cases undoubtedly belong to the group described by Marie and Bénisty. Immediately following a gunshot wound in the neck the injured man falls paralyzed in

all extremities. Within a few days function is recovered in one arm and leg, leaving a spastic paralysis of the lower extremity on the side of the injury and a flaccid paralysis in the upper

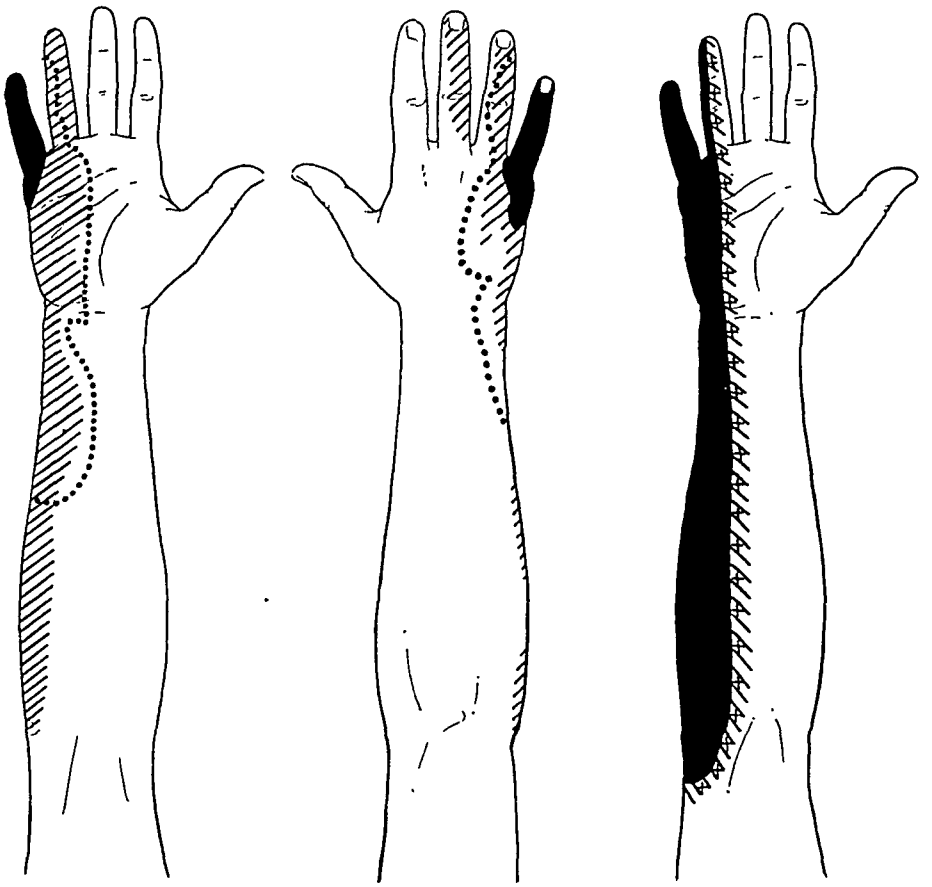


FIG. 251. Sensory loss in lesions of cervical eighth and dorsal first roots.

extremity, with a loss of sensation to pain and temperature sense on the opposite side of the body, below the level of the lesion. The paralysis in the leg spontaneously and often rapidly disappears, leaving a long lasting flaccid paralysis in the upper extremity which is due to a lesion of the brachial plexus. Often the wound of entrance was about the face, mouth or jaws and at times there occurred a paralysis of the

seventh or twelfth cranial nerves. A paralysis of the cervical sympathetic was also common.

We have the records of only ten such cases, although we

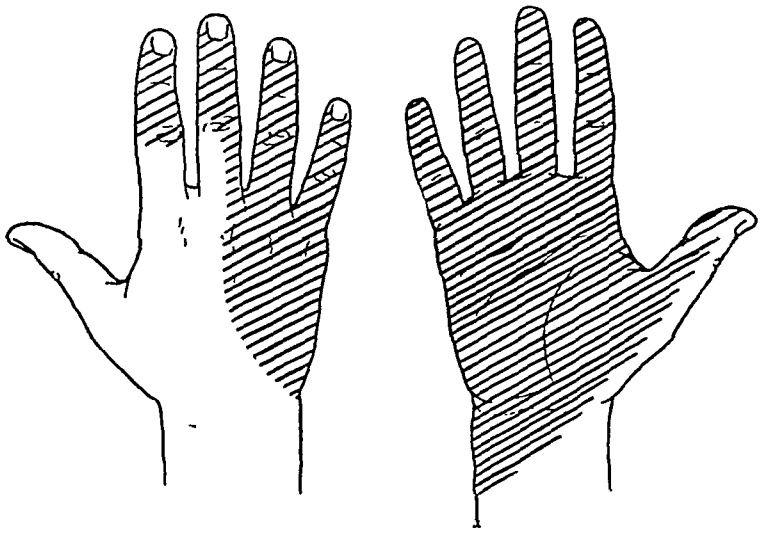


FIG. 252. Sensory loss in ulnar and median nerve lesion.

remember seeing a number of others. They exhibited a similar symptomatology and course and a description of one will suffice.

V. E. N., Pvt., 26th Infantry, was wounded July 18, 1918. A gunshot wound had been received at the angle of the inferior maxilla with the exit of the bullet wound over the second cervical spinous process. He felt the missile strike him and was thrown into the air. At first he had tonic and then a few clonic jerks in his extremities which were then paralyzed. He began to use his right leg two days later, then his right arm, and shortly after he regained some power in his left leg. When admitted to U. S. B. H. No. 13, on September 19, 1918, he was able to perform all movements with the right lower extremity with good power. There was moderate weakness in the muscles of the left lywer extremity, the flexors of the legs being the weakest muscles. All of the muscles of the right upper extremity were very weak. There was a paralysis of all of the muscles of the left upper extremity, with the exception of the deltoid, biceps, brachialis anticus, latissimus dorsi, pectorals, levator anguli scapulae and serratus magnus, which were very weak. The deep reflexes in the lower extremities were increased and were greater on the left side. In the upper extremities they were absent,

with the exception of the biceps, which was brisk on the left and normal on the right. The superficial reflexes were absent on the left side and a Babinski reflex was present. There was a Horner's syndrome on the left

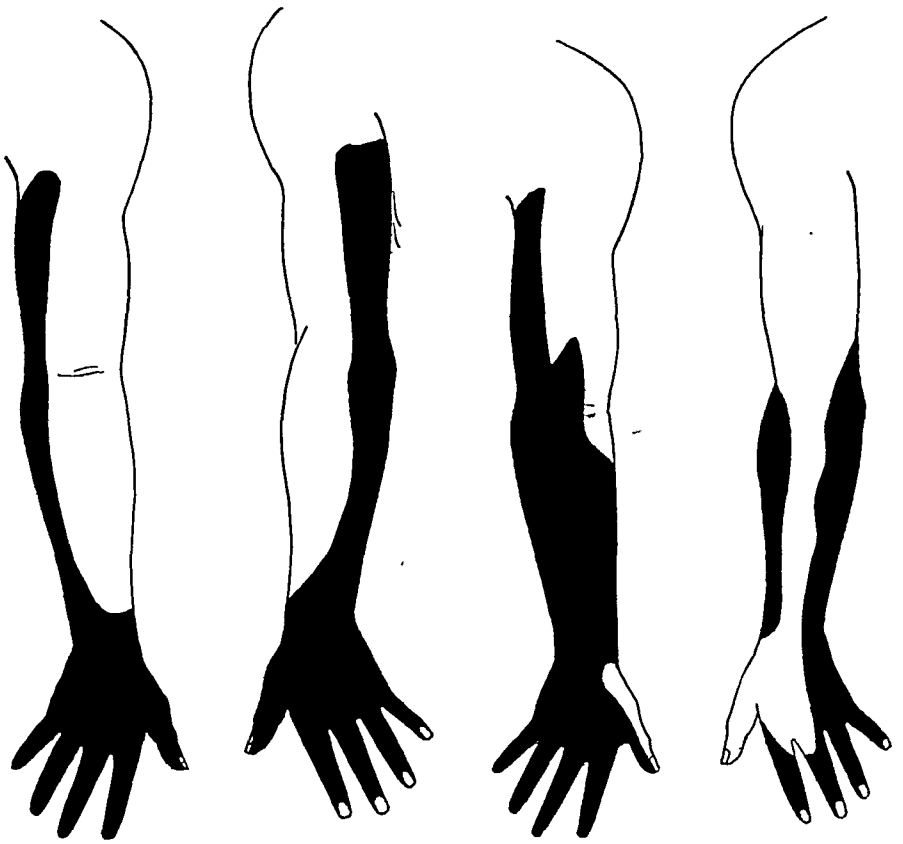


FIG. 253. FIG. 254.

FIG. 253. Sensory loss in lesion of ulnar, median, radial and medial cutaneous nerves of arm and forearm.

FIG. 254. Sensory loss in lesion of ulnar, median, musculocutaneous and medial cutaneous nerves of arm and forearm.

side. There was an hyperesthesia of the left side of the body below the second dorsal segment. There was an analgesia and a loss of temperature sense on the right side distal to the sixth cervical segment, and loss of sensation over the fifth, sixth, seventh and eighth cervical segments on the left.

Particularly interesting are such cases in which the upper level of the contralateral analgesia is a number of segments

below the residual sensory loss on the side of the lesion (Fig. 260). It has been assumed from a study of Brown-Séquard paralysis that the fibers for pain and temperature senses

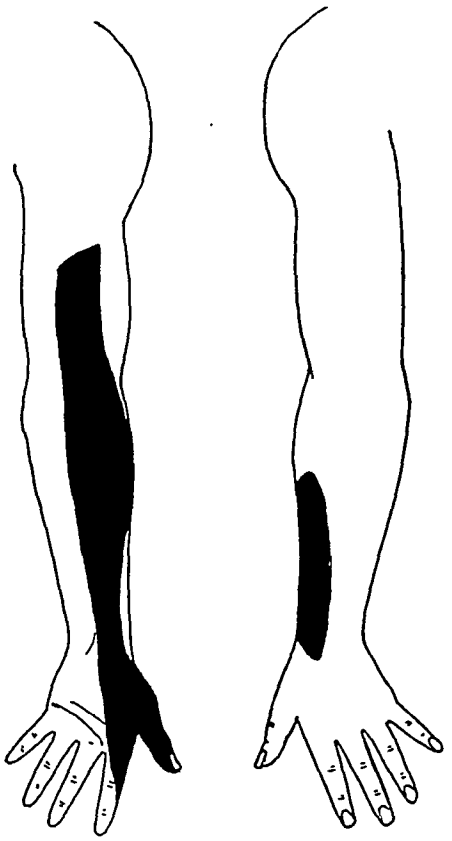


FIG. 255. Analgesia of thumb and index finger when pronator radii teres is paralyzed.

require a number of segments for complete crossing in the cervical cord. The observation that these fibers cross more directly in the dorsal region points out the possibility that the sensory loss which results from a brachial plexus lesion may be misinterpreted as that due to a cord lesion. It is possible that the absence of a thoracic plexus has led to a more accurate interpretation of the crossing of protopathic fibers in this locality.

Anatomy

The muscles of the extremities may receive their innervation from more than one segment of the spinal cord. This

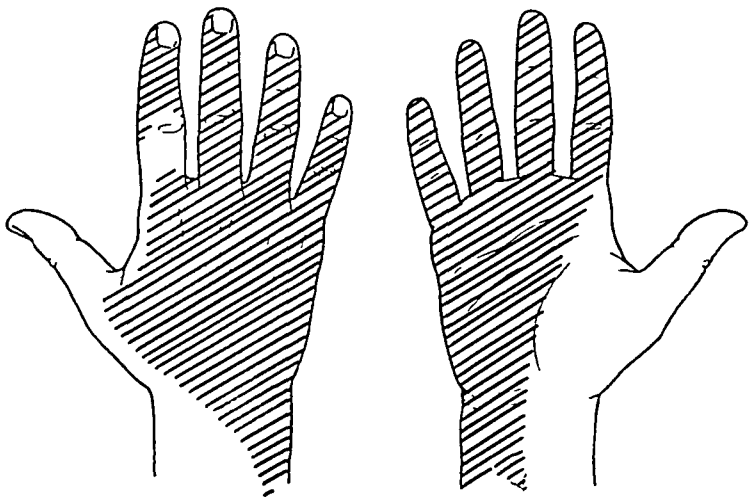


Fig. 256. Sensory loss in lesion of inner primary cord of brachial plexus.

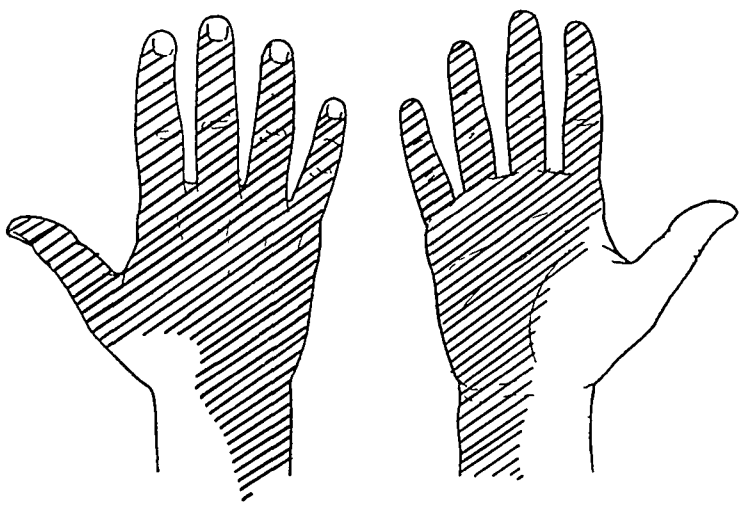


Fig. 257. Sensory loss in lesion of posterior and inner cords of brachial plexus.

anatomical fact accounts for the development of nerve plexuses for the upper and lower extremities.

The brachial plexus consists of the anterior branches of the spinal nerves which are distributed to the muscles and skin of the shoulder girdle and upper extremity. The fifth, sixth,

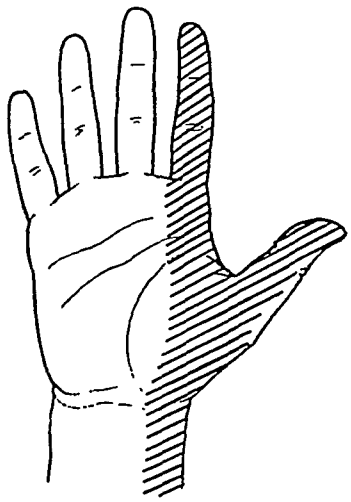


FIG. 258. Sensory representation of outer head of median nerve.

seventh and eighth cervical and the first thoracic spinal nerves form the brachial plexus. There may be variations in this arrangement of the plexus and the two most common forms are those which occur in the pre-fixed and post-fixed types. In the former the plexus receives a large contribution from the fourth cervical spinal nerve. In the post-fixed type the second dorsal spinal nerve contributes to the formation of the plexus. A difference in the type of plexus in the two upper extremities may exist in the same individual.

The plexus is situated in the posterior triangle of the neck and lies upon the dome of the pleura. It lies behind the clavicle and gives rise to its terminal branches within the axilla. Roughly, the plexus has the form of two triangles apposed to their apices. The base of the superior triangle lies against the vertebral column. Its external and superior side has an oblique direction downward and externally. The inferior side lies upon the superior orifice of the thorax and the dome of the pleura. The inferior triangle is smaller and is less regular in shape.

It lies within the axilla and its base corresponds to the origin of its terminal branches.

The anterior branch of the fifth cervical spinal nerve unites

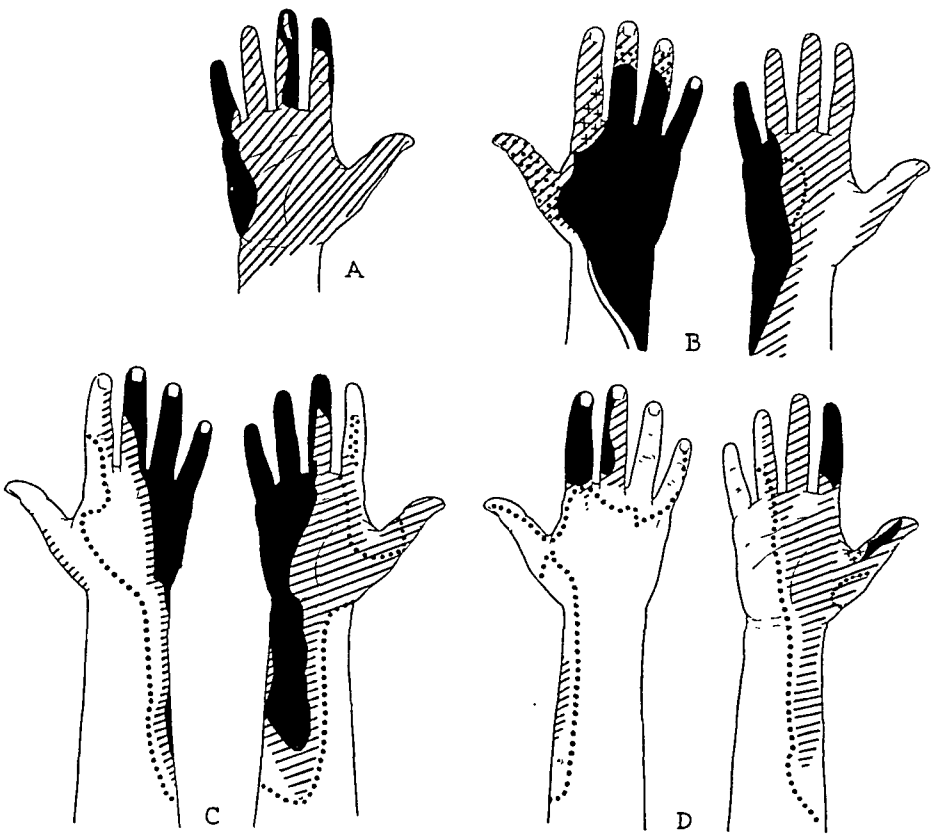


FIG. 259. Sensory loss in recovering lesions of brachial plexus.

A. Patch of return of pain in isolated supply; recovery between adjacent nerves.

B. Recovery of pain, not touch.

C. Interlacing of loss of cold; return of touch in isolated supply; recovery of touch and not cold.

D. Recovery of touch, not cold; patchy return of touch.

at an acute angle with the anterior branch of the sixth cervical spinal nerve to form the *upper primary trunk* of the plexus. The anterior branches of the eighth cervical and first thoracic spinal nerves unite to form the *lower primary trunk*. The anterior branch of the seventh cervical nerve lies between these two trunks and forms the *middle primary trunk*. Each primary

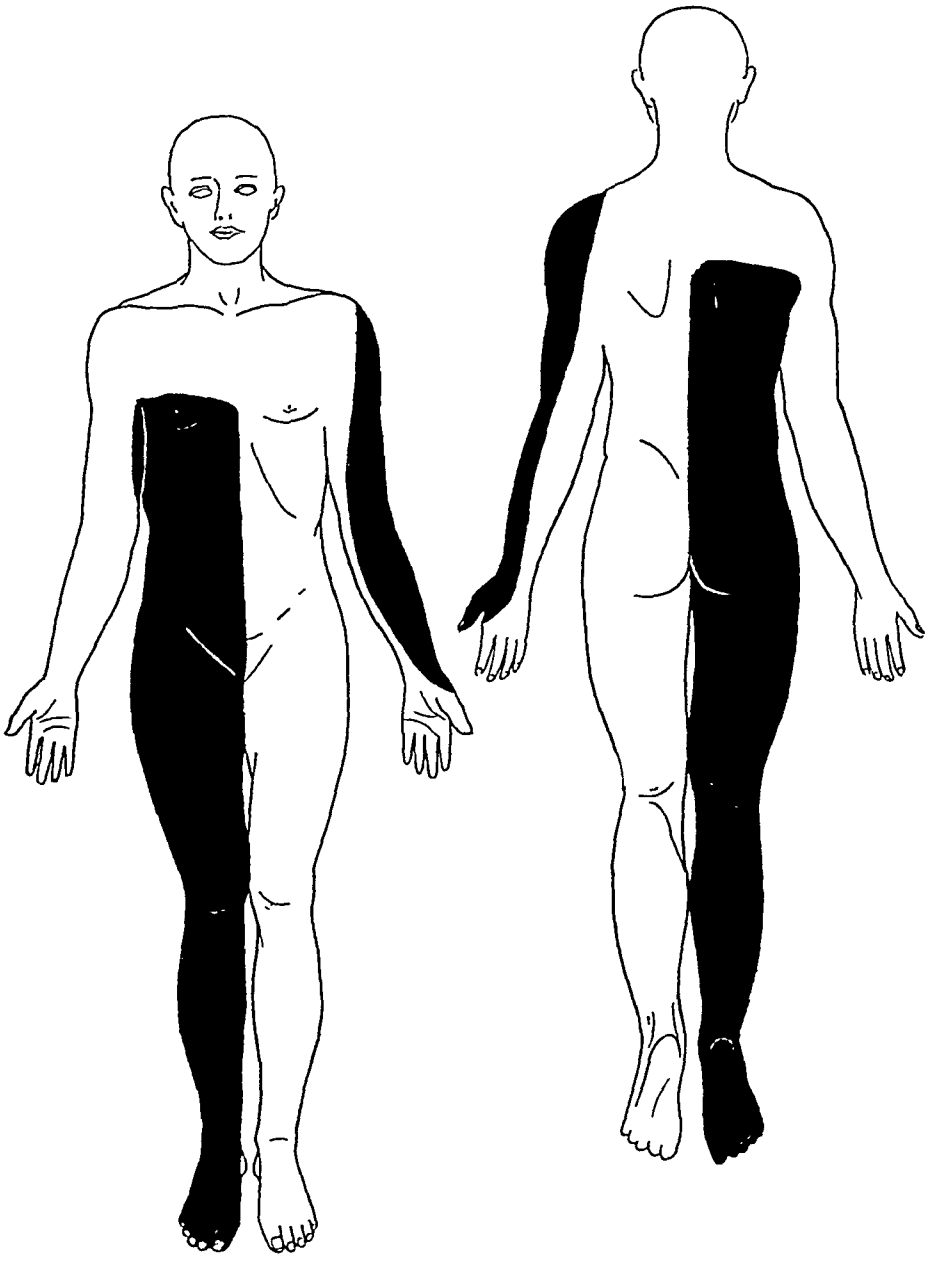


FIG. 260. Sensory loss in Brown-Séquard lesion of spinal cord with ipsilateral brachial plexus injury.

trunk divides into an anterior and posterior division. The anterior divisions of the upper and middle primary trunks unite to form the *lateral cord* of the brachial plexus. The anterior divisions of the lower primary trunk form the *medial cord* and the posterior divisions of each of the primary trunks unite to form the *posterior cord* of the plexus. The lateral cord gives rise to the lateral head of the median and the musculocutaneous nerves. The medial cord gives off as its terminal branches the ulnar, medial head of the median, medial cutaneous nerve to the arm and the medial cutaneous nerve to the forearm. The radial and axillary nerves are the terminal branches of the posterior cord of the plexus (Fig. 261).

Because of the curve of the vertebral column the roots of the fifth, sixth and seventh cervical nerves are placed more anteriorly than are those of the eighth cervical and first thoracic nerves. Leaving the transverse processes of the vertebrae, the roots pass obliquely downward and externally to enter between the anterior and middle scaleni muscles. It is not uncommon to see the fifth and sixth cervical roots pierce the anterior scalenus muscle to lie upon its anterior surface. The fifth and sixth roots unite to form the primary trunk on the scalenus medius external to the outer border of the anterior scalenus. This point is external to the outer border of the sternomastoid muscle about 2 cm. above the clavicle and is known as Erb's point. When the fourth cervical contributes to the brachial plexus, the anastomosis occurs above that point.

The eighth cervical and first thoracic roots have a much more complicated anatomical arrangement. The anterior branch of the eighth cervical and that of the first thoracic appear in the depth of a small fossa bounded externally by the transverse pleural ligament; internally, by the vertebropleural ligament; below, by the sloping dome of the pleura; and anteriorly, by the posterior extremity of the first two ribs and the vertebral column. The eighth cervical runs obliquely forward and externally to the superior surface of the

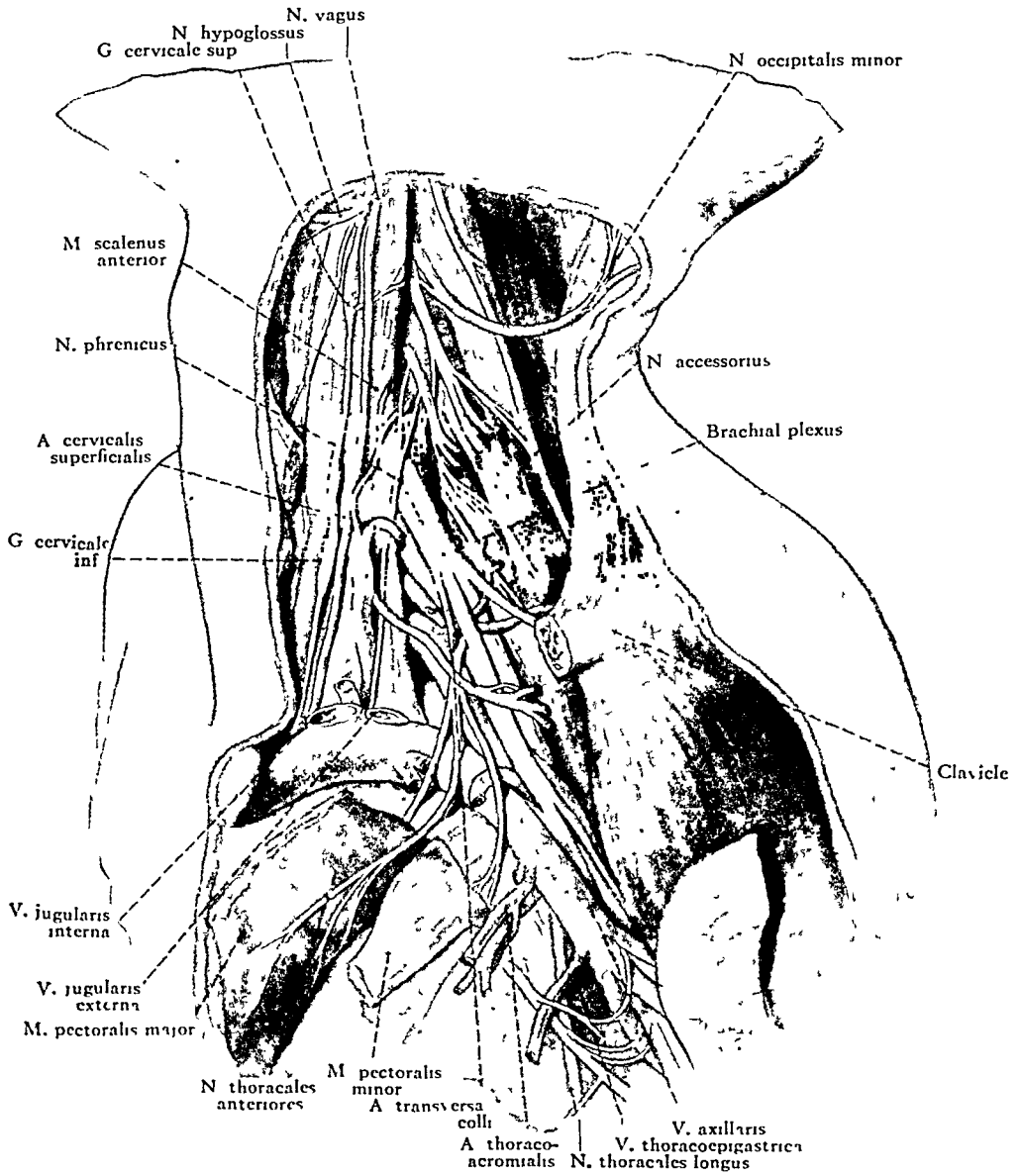


FIG. 261. Anatomy of brachial plexus.

neck of the first rib. The transverse spine of the seventh cervical vertebra overhangs it and it is hidden in front by the inferior cervical sympathetic ganglion. As it passes more



FIG. 262. Incisions to expose brachial plexus.

externally it leaves the rib and the sympathetic ganglia and overlies the dome of the pleura. It is hidden behind the transverse pleural ligament and at the inner border of the posterior scalenus muscle it unites with the anterior branch of the first thoracic nerve. The latter appears below the neck of the first rib, occasionally it is in contact with it, and passes forward and externally to cross the internal border of the rib. The costopleural ligament lies over the root which is in contact with the dome of the pleura. As it passes from beneath the ligament it unites with the eighth cervical. The contribution of the second thoracic to the plexus, when it is present, is usually very slender.

Beneath the clavicle the primary trunks give rise to the anterior and posterior divisions, which, as has been pointed out, form the cords of the plexus. The nerves lie upon the middle and posterior scalenus muscles and are bound to them by a very strong aponeurosis. The plexus is crossed obliquely by the omohyoid muscle and may be said to occupy the two

triangles of the neck which are divided by this muscle. The large part of the plexus is found in the inferior, or omoclavicular, triangle. The fifth, sixth, seventh cervical roots and the upper primary trunk occupy the omotrapezial triangle. The inferior primary trunk lies deeply upon the first rib in the space between the anterior and middle scaleni, and is difficult to see without dividing the clavicle. It is in intimate relation with the subclavian artery which in general lies anterior and below this part of the plexus.

The superficial transverse cervical artery appears at the external border of the scalenus anticus muscle. It passes upward and externally to the anterior border of the trapezius. In so doing it crosses the upper and middle primary trunks. The posterior scapular artery appears between the scaleni, passes posteriorly and externally and after a short ascending course becomes quite tortuous. It lies anterior to the middle primary trunk and finally passes between it and the upper primary trunk.

Superficially, the plexus is hidden by the superficial and middle layers of the cervical aponeurosis and the fibers of the platysma. The external jugular vein, as it appears at the posterior edge of the sternomastoid, is separated from the plexus by these layers of fascia but after perforating them at the sternoclavicular angle, it runs medially toward the subclavian and there lies close to the inferior primary trunk.

Below the clavicle and in the apex of the axillary space the lateral, medial and posterior cords are formed. They are intimately related to the axillary artery which they separate from the axillary vein. Externally lie the lymphatic chain and glands which drain the upper extremity. Just below its superior edge and posterior to the pectoralis minor muscle the cords of the plexus give rise to their terminal branches. Each of these nerves will be described separately in detail.

As has been stated the fourth cervical and second thoracic spinal nerves may have a branch of anastomosis with the plexus. In addition there are anastomoses with the sympa-

thetic ganglia in the cervical region. To each of the cervical spinal nerves a grey ramus communicans runs from the sympathetic chain.

Certain collateral branches arise from the constituent roots of the plexus before the lateral, medial and posterior cords are formed. These may be divided for convenience into those supplying the muscles on the anterior and posterior aspects of the shoulder girdle. There are six posterior branches. The *dorsalis scapulae* (nerve to the levator anguli scapulae and the rhomboids) and the *long thoracic* (nerve to the serratus anterior) arise from the posterior aspect of the roots. The *upper and lower subscapular nerves,* the *thoracodorsalis* (nerve to the latissimus dorsi) and the *nerve to the teres major muscle* arise from the posterior surface of the primary trunks.

The *dorsalis scapulae* nerve arises from the fourth and fifth cervical roots. At first it rests upon the scalenus medius to which it may send a twig, and then it passes obliquely down and externally between that muscle and the posterior scalenus. It is applied to the anterior surface of the levator angulae scapulae muscle to which it sends two or more twigs. Curving about the inferior border of the muscle the nerve lies between the small serratus superior in front and the rhomboids behind. It descends upon the anterior surface of the latter near the spinal border of the scapula. The terminal twigs supply the rhomboids throughout.

The *long thoracic* nerve arises by two or three roots. The first comes from the fifth cervical and occasionally from a trunk common to the dorsalis scapulae. The second and third roots come from the sixth and seventh cervical roots. The first two are quickly united and pierce the middle scalenus muscle. The lower root joins the nerve after it has already given off a twig to the upper digitations of the serratus anterior. The nerve passes through the axilla behind the branches of the brachial plexus which it separates from the vessels. It descends vertically upon the superficial surface of the serratus anterior about 3 to 4 cm. behind the line of emergence of the lateral

perforating branches of the intercostal nerves. The external mammary artery accompanied by a lymphatic chain lies between the nerve and these perforating branches. The nerve gives off twigs to the serratus anterior muscle along its posterior border. Certain digitations, especially the superior ones, may receive more than one twig of supply.

The *upper subscapular nerve* arises from the upper trunk of the plexus and therefore receives fibers from the fifth and sixth cervical spinal nerves. Soon after its origin it follows the external border of the upper cord and then courses inferiorly and internally along the spinal border of the scapula. Its twigs are distributed to the upper half of the subscapular muscle. The *lower subscapular nerve* may be given off directly from the posterior cord of the plexus or it may arise by a common trunk with the axillary. The nerve descends upon the anterior surface of the subscapular muscle external to the upper subscapular. It is distributed to the lower fibers of the subscapular muscle.

The *nerve to the teres major* muscle has a variable origin. It may arise directly from the posterior cord or from a common trunk with the axillary. The nerve courses inferiorly and medially and crosses the inferior and external border of the subscapular muscle to which it gives a few twigs. It may cross the inferior scapular artery to reach the teres major at the external border of the scapula. It divides into a variable number of twigs, which pierce the anterior surface of the muscle and supply it along its entire length.

The *thoracodorsalis* nerve arises directly from the posterior cord of the plexus. It receives fibers from the sixth, seventh and eighth cervical spinal nerves. It descends upon the subscapular muscle external to the nerve to the teres major. The nerve borders the anterior surface of the latissimus dorsi in the axilla and then gives off collateral twigs to supply it throughout its extent.

There are four collateral branches which supply the muscles on the anterior aspect of the shoulder girdle. The *supra-*

scapular nerve arises from the lateral cord of the brachial plexus and therefore receives fibers from the fifth and sixth cervical roots. It lies between the lateral cord and the long thoracic nerve. This nerve is one of the largest of the collateral branches. It crosses the first digitation of the serratus anterior and behind the clavicle it passes laterally around the pectoralis minor together with the suprascapular artery, to enter the suprascapular notch. The nerve is distributed to the supraspinatus and infraspinatus muscles. The *nerve to the subclavius muscle* arises from the upper primary trunk and receives fibers from the fifth cervical constantly and occasionally from the sixth. The nerve crosses the external edge of the anterior scalenus muscle to reach the subclavius at the junction of its middle and inner thirds. In its course it is crossed by the superficial transverse cervical and the posterior scapular arteries, and is hidden by the posterior border of the sternomastoid. It gives off an important branch of anastomosis to the phrenic nerve. The *external anterior thoracic nerve* almost constantly has two parts which arise from the upper primary trunk and from the middle primary trunk. It receives fibers from the fifth, sixth and seventh cervical spinal nerves. Both parts of the nerve descend in front of the axillary artery. They pierce the clavicular-pectoral aponeurosis and are distributed to the under surface of the pectoralis major. The superior one of the divisions supplies the clavicular fibers while the inferior trunk innervates the sternal fibers. The *internal anterior thoracic nerve* arises from the lower primary trunk. It receives fibers from the eighth cervical and first thoracic spinal nerves. It descends behind the axillary artery which it crosses just external to the origin of the external mammary. It receives an anastomosis from the external anterior thoracic nerve. It is distributed to the under surface of the pectoralis minor muscle.

Physiology

The physiologic action of the muscles supplied by the terminal branches will be described under each of those nerves

in detail. The muscles innervated by the collateral branches of the plexus are:

The *levator anguli scapulae* muscle passes from the superior angle of the scapula to the transverse process of the first four or five cervical vertebrae. It raises and rotates inward the superior angle of the scapula. The *rhomboid muscles* pull the scapula upward and toward the median line. This approximates its inferior angle to the vertebral column, and steadies the vertebral border of the scapula. Thus it is easier to draw the arm backward and outward.

Contraction of all of the fibers of the *serratus anterior* muscle carry the scapula forward, outward and upward. It is an important accessory muscle of respiration. It contracts with the pectoralis major to push forward with the shoulder. It assists the deltoid in raising the arm from the side. The *subscapularis* muscle rotates the arm inward. The *teres major* raises the shoulder, brings the inner aspect of the arm and the axillary border of the scapula together and draws the humerus backward. The *latissimus dorsi* muscle depresses the shoulder, draws the arm inward, rotates it backward and brings the scapula to the midline. When both latissimus dorsi muscles contract simultaneously the shoulders become less prominent and are carried obliquely backward and inward.

The *supraspinatus* muscle lifts the humerus and draws it forward and outward. Its action is strong enough to raise the arm when the deltoid is paralyzed. The *infraspinatus* rotates the head of the humerus outward. The *pectoralis major* muscle adducts and draws the arm forward when it is fully contracted. The upper part of the muscle draws the point of the shoulder upward and forward so that if the arm is raised to the vertical, contraction of this portion of the muscle draws the shoulder forward and toward the midline. It also depresses it to the horizontal and in this action is aided by the lower portion of the muscle. The *pectoralis minor* draws the shoulder forward. It assists the serratus anterior muscle in the performance of this action. The *subclavius muscle* depresses the clavicle. If the

shoulder girdle is fixed it is capable of raising and fixing the first rib in inspiration.

Surgery

Traumatic lesions of the brachial plexus are common in civil and military life. They may be produced directly by blows upon the side of the neck or shoulder, or by penetrating wounds; or indirectly by stretching or avulsion of the roots of the plexus. The mechanism of these indirect injuries is the same in adults or infants in whom they are encountered most frequently as birth palsies. The mechanism consists of depression of the shoulder while the head and neck are turned to the opposite side. This increases the angle between the shoulder and neck. If the injury is severe the upper roots may be avulsed completely. This occurs usually external to the intervertebral foramina. The upper trunks may be overstretched so that the nerve sheath remains intact. In such cases spontaneous regeneration might occur more often were it not for certain anatomical facts. The prevertebral layers of the deep cervical fascia covers the plexus closely and hemorrhage beneath this fascia may be responsible for dense scar tissue which produces serious compression. The relation of the plexus to the apex of the lung and the important vessels in the neck make penetrating wounds particularly dangerous. Combined lesions of the axillary artery or its branches and the plexus may be characterized by an hematoma and subsequent scar tissue formation. Posttraumatic aneurysms are not infrequent complications of an injury to the plexus low in the neck. All of these possible lesions of associated structures seriously complicate the surgical treatment of plexus lesions.

The results of the surgical treatment of severe plexus lesions are disappointing. This is due to the location of the lesion within or near the intervertebral foramina which makes it surgically unapproachable or to involvement of the cord when the roots are avulsed within the spinal canal. If the roots are divided they must be repaired with the closest regard to the approximation according to the original anatomical and

physiological pattern. Even though regeneration of nerve fibers may occur the function which results from a distortion of nerve pattern may make an arm useless. In many of the less severe lesions a careful, painstaking neurolysis will produce a surprisingly good result in function.

The entire brachial plexus may be exposed by an incision which consists of three parts (Fig. 264). The first is the longitudinal incision which parallels the posterior border of the sternomastoid muscle. The second portion is placed horizontally parallel to the clavicle and the third portion is a continuation of the latter incision over the anterior aspect of the shoulder to the medial side of the coracoid process and extends along the medial border of the biceps tendon to below the insertion of the pectoralis major. Any one of these three incisions may be used to expose the upper or lower parts of the plexus or they may be combined to expose it in its entirety.

The prevertebral layer of the cervical fascia should be exposed and the transverse cervical vessels ligated. The fascia is divided and the lateral border of the scalenus anticus muscle should be identified and exposed as it lies beneath the sternomastoid. The upper trunk appears from the lateral border of the scalenus anticus and lies upon the scalenus medius. If the lesion is lower down, it may be necessary to divide the clavicle. Holes should be drilled through the clavicle to facilitate its later approximation with wire. A Gigli saw should be used to divide it subperiosteally. To obtain a complete exposure of the subclavicular portion of the plexus it may be necessary to divide the insertion of the pectoralis major muscle from the humerus and the pectoralis minor from the coracoid process. Division of the subpectoral fascia will expose the nerves and vessels and by retracting the divided ends of the clavicle upward the cords of the plexus may be followed upward or their terminal branches may be followed downward. Care must be taken to preserve the anterior thoracic nerves which supply the pectoral muscles. Occasionally, direct injury to the axillary artery or compression by scar tissue may reduce it to the appearance of a fibrous cord.

Chapter XXX

THE SCIATIC NERVE

It is difficult to determine the frequency of injuries to the sciatic nerve because often when one or the other of its terminal divisions is either injured alone, or predominately injured, the condition is classified as peroneal or tibial nerve injury. From the material of Mann, Gundermann, Thoele, Spielmeyer and Reichmann, the sciatic was injured in 8.18 per cent; the peroneal in 12.28 per cent and the tibial in 2.4 per cent. Roeper and Wexberg reported 33 per cent, including the branches; and Toby Cohn found the sciatic or its branches injured in 122 cases in a group of 513 cases. The sciatic trunk was injured in 51 of these; the peroneal in 29; the tibial in 26; and both in 15 cases.

In our material, both branches of the sciatic were injured in 160 cases; the peroneal branch above the bifurcation in 11; and the tibial in 2 cases. Below the bifurcation the peroneal was injured in 120 cases; the tibial in 25; the anterior tibial in 16; and the posterior tibial in 16 cases.

In addition to gunshot and stab wounds, injury of the sciatic nerve may result from fracture of the femur. This may occur immediately as the result of direct injury, or later from callus formation. It occurs as the result of reduction of dislocations of the hip. It occurs in infants as the result of injury at birth. Jumping upon the extended foot, and sudden, unexpected extension of the lower extremity with flexion of the trunk, has produced injury to the sciatic nerve. Saddlers and plasterers are predisposed to injury of the sciatic. At times cases have been observed following operative anesthesia, and it has been injured at operation. It has frequently been injured by the injection of arsphenamine, ether, alcohol and mercury. Alcoholic polyneuritis chiefly affects the sciatic nerves. They are, of course, involved in other polyneuritides, such as lead,

arsenic and those due to infectious diseases. The sciatic may be the seat of a mononeuritis as the result of an osteoarthritis of the spine or sacro-iliac joint.



FIG. 263. Deformities seen in sciatic nerve paralysis.

MOTOR SYMPTOMS

Because the nerve to the semitendinosus and membranosus is given off very high, a complete paralysis of flexion of the leg is rare. As a rule, when the patient is examined in the prone

position some weakness may be made out in the biceps, and to a lesser degree in the other two muscles.

While standing, the foot dangles and drops. In walking the,

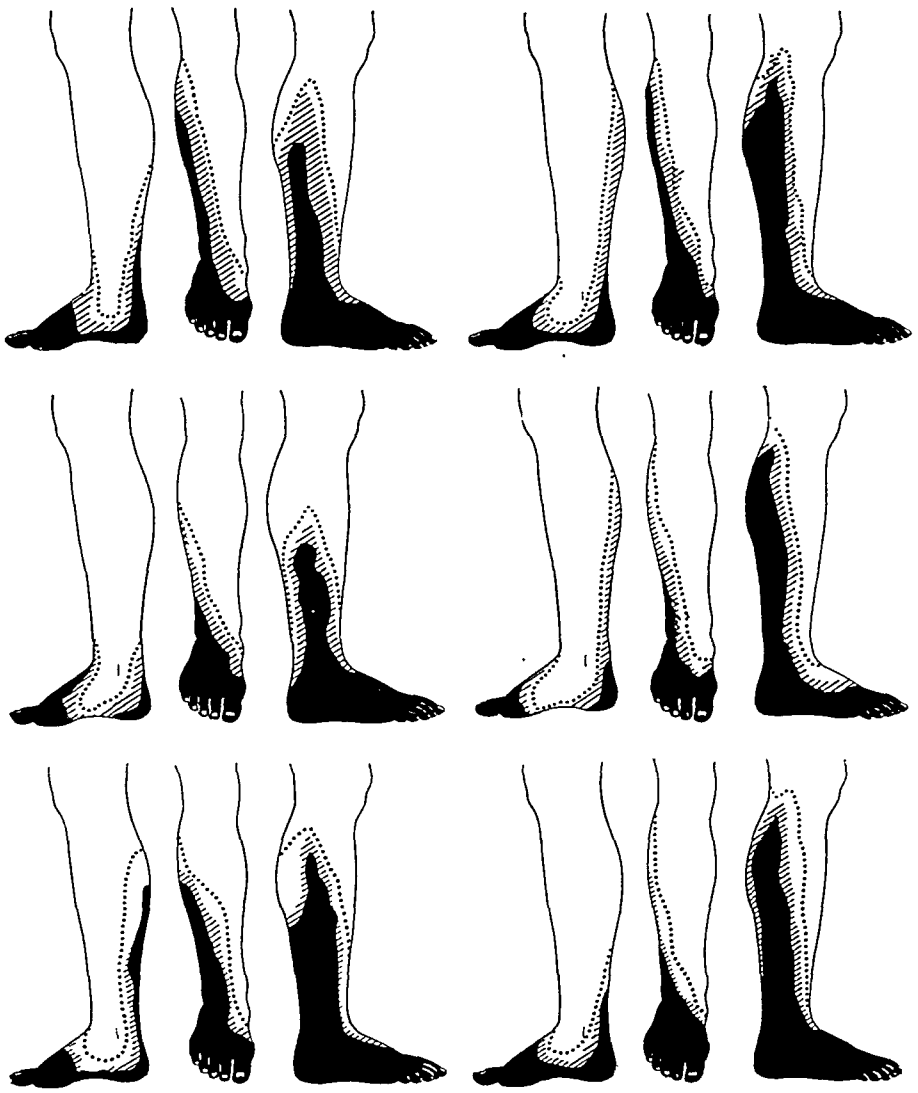


FIG. 264A.

FIG. 264. Sensory loss in sciatic nerve lesions. A. Sciatic.

knee at times is carried too high and the foot is brought down somewhat as in the steppage gait of a peroneal nerve palsy.

As time goes on, fibrosis and shortening of ligaments produces a limitation of movement of the foot and the patient may walk with relatively little disability, using the paralyzed

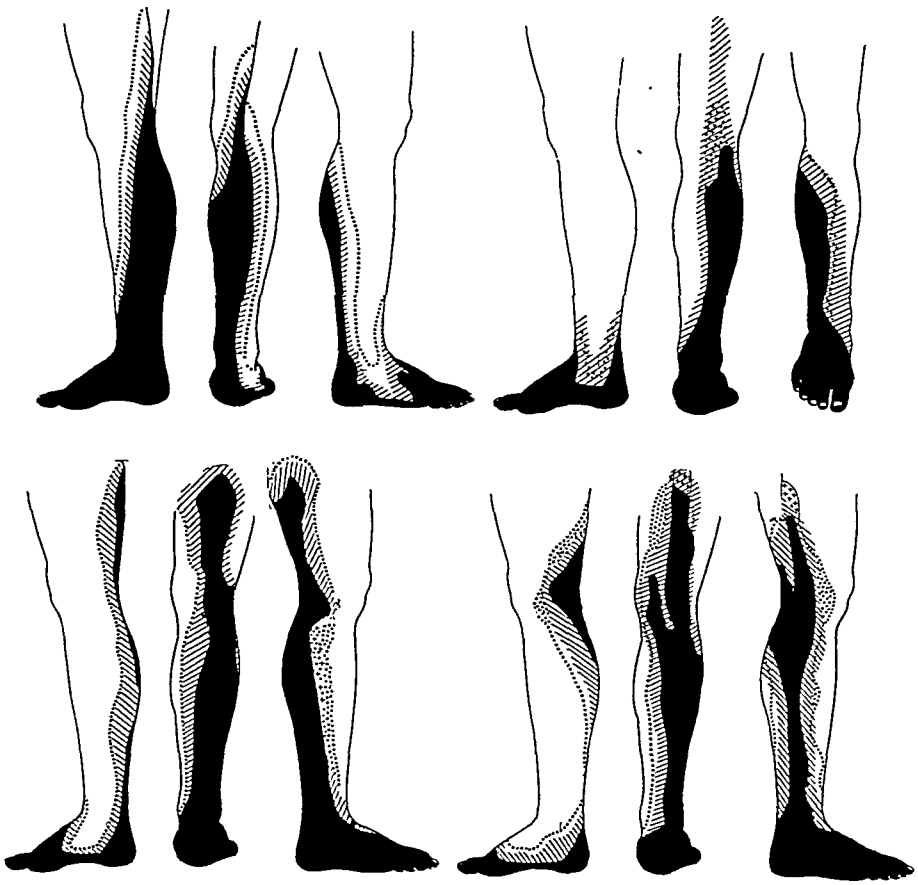


FIG. 264B. Sciatic and small sciatic.

extremity much as a peg leg. All of the muscles below the knee are paralyzed and the patient cannot stand on his heels or toes. Running is impossible. (Fig. 263.)

Atrophy of the muscles is often concealed by the edema and infiltration of the tissues of the leg. When it is observable, in some cases pronounced atrophy of all of the muscles may be seen. The knee jerk is always present and the Achilles jerk is always absent. When the plantar reflex is sought for by

stimulating the inner side of the sole, contraction may occur in the fascia lata, but no movement is seen in the foot.

SUPPLEMENTARY MOVEMENTS: Interpretation of muscle



FIG. 265. Isolated supply to pain in a sciatic nerve lesion.

function in the lower extremity is much simpler than that in the upper, chiefly because of the minor importance of the intrinsic foot muscles as compared with those of the hand. Attention has been directed chiefly toward the movements of the larger muscles in the lower extremity. Inasmuch as such supplementary movements can occur only when one of the divisions of the sciatic nerve is totally paralyzed and the other partially or not at all involved, description of these movements will be made under the heads of the peroneal and the tibial nerves.

SENSORY SYMPTOMS

Objective sensibility is diminished or lost over the entire foot, with the exception of the inner border of the arch and the internal malleolus. It is lost over the dorsum of the foot, over both sides of the heel and the outer side of the leg to the knee (Fig. 264).

The overlap of adjacent uninjured nerves is considerable, so that the upper level of the loss of pin prick may extend only to the middle third of the leg. Above this only epicritic sensibility was found lost over the accepted sensory distribution of the nerve. The overlap to deep sensibility elicited by an algometer was greater than to pain, and pressure pain could be felt above the ankles. It is noteworthy that we could never elicit sensation by pinching the toes in a case of complete section of the sciatic nerve (Fig. 265).

In complete lesions subjective sensory disturbances are usually negligible, except for a very short time following injury. The sensory loss contributes largely to the disability in producing unsteadiness of gait.

Vasomotor and Trophic Changes

As the result of ankylosis, injury to the sole is common. Vasomotor changes, and perhaps some trophic disturbance which interferes with the healing, make ulcerations common. Often an ulcer may follow some slight abrasion beneath the ball of the great toe. It may progress to a fully developed perforating ulcer which involves the joint.

Edema of the leg and foot is common, and brawny infiltration of the calf is seen. The foot is often purplish in color, warmer than normal and has a distinctly dry skin.

Plantar hyperkeratosis is of frequent occurrence. The satin-like skin over the dorsum of the foot is often covered by scales and at times crusts. Sweating is diminished except over the inner border of the foot and the internal surface of the leg. The ball of the foot is frequently more prominent, and the toes may be hyperextended at the first phalanges. (Fig. 266.)

Partial Lesions

Dissociated or partial paralysis may be due to a greater damage to either the tibial or the peroneal branches, or to partial injury of both. Operative and clinical experience shows that the peroneal suffers greater damage, as a rule, than the

tibial. Of a series of 37 cases seen soon after injury, the muscles supplied by the peroneal were paralyzed and those supplied by the tibial were weak in 12 cases. All of the muscles were



FIG. 266. Trophic disturbances in sciatic nerve lesions.

weak in 10 cases. The tibial group were paralyzed and the peroneal group were weak in 4 cases. The peroneal nerve alone

was paralyzed in 2 and the tibial nerve alone in 2 cases. The extensor muscles of the toes were paralyzed in 2 cases and all of the muscles were paralyzed in 2 cases.

Signs of Motor Recovery

According to Bénisty, the muscles which are first to recover are, according to the individual case, the tibialis anticus, the peroneus longus or the gastrocnemius. Much more rarely recovery begins in the tibialis posticus. The flexors and extensors of the toes are always the last to recover.

The Medical Research Council of Great Britain reported that, as a rule, the tibial nerve showed signs of return of function earlier than did the peroneal. Stopford's records of sutured cases showed that the order of recovery is the gastrocnemius, tibialis anticus, extensor longus digitorum and extensor proprius hallucis.

The records of the Military Orthopedic Hospital, Shepherd's Bush, gives the order of recovery in 8 cases as tibialis posticus, gastrocnemius, tibialis anticus, extensor longus digitorum, extensor longus hallucis, extensor brevis digitorum and peroneus longus.

In a group of cases in the American material it was found that in 41 recovered cases there was a return of function in the tibialis anticus in 18, in the gastrocnemius in 11; in all muscles in 9; in the tibialis posticus in 8; in the peronei in 4; in all muscles supplied by the peroneal in 3; in the flexors of the toes in one; and in all muscles supplied by the tibial in one case.

In 14 cases following suture, function returned first in the gastrocnemius in 8 cases; in the tibialis posticus in 5 cases; in the peronei in 4 cases; in the flexors of the toes in 3 cases; in the tibialis anticus in 3 cases; and in the extensors of the toes in one case.

In severe cases and following suture, return of function in the branches of the tibial occurred earlier than in those of the peroneal. Extension of the toes was very late in appearing,

and in spontaneously recovering lesions flexion of the toes occurred very late.

Signs of Sensory Recovery

Sciatic nerve lesions which recovered spontaneously occasionally showed marked sensory loss with good motor recovery. Recovery of pain sense only in the areas supplied either by the tibial or peroneal nerves occurred frequently. When sensation was returning to the sole, areas of recovery to touch and pain with loss to cold sense were seen. Areas of recovery to touch and not to cold were also observed over the leg. When sensation had partly returned in the area supplied by the tibial, the area of analgesia in the region supplied by the peroneal occupied a wider area than is seen in isolated lesions of the peroneal. Recovery of touch and pain in patchy and indented patterns occurred. (Fig. 267.)

Generally in lesions which recovered following operation, the loss of pain was more extensive. When partial recovery had taken place in the areas supplied by the tibial, the sensory loss produced by the lesion of the peroneal was very extensive. When one or the other divisions of the sciatic showed unequal regeneration, this condition was found to be the result of a lesser injury to the nerve which showed signs of first recovery. (Figs. 268.)

Painful Lesions

As was noted in the case of the median nerve, so in the sciatic nerve some injuries result in a painful type of paralysis. It has been found that only those injuries are painful which show an injury to the fibers of the tibial nerve.

During the first week complete paralysis is often present. Then motor recovery begins, most often in the region of the tibial. In some cases, however, when the peroneal branch is less injured, movements controlled by this nerve are the first to recover. As with the median nerve, involuntary movements

are sometimes observed, consisting of abduction and adduction of the foot, or flexion of the toes.

The pain is referred to the sole and at times is extremely



FIG. 267. Sensory loss in spontaneously recovering lesions of sciatic nerve.

A. Extensive sensory loss with marked motor recovery.

B. Recovery of touch but not of pain, and interlacing of cold sense

C. Recovery of pain sense in tibial distribution with wide area of loss of pain sense in peroneal area.

D Recovery of touch and pain but not of cold sense; patchy recovery of touch sense.

E Complete recovery of peroneal and of pain sense in tibial areas.

F. Recovery of pain sense in isolated supply and cold sense in patches.

G. Recovery of touch sense in patchy areas but no return of cold sense.

H Patchy recovery of pain sense in tibial; recovery of peroneal.

severe. Burning pain is characteristically present and is associated with a spasm of the muscles in the sole. The calf may be exquisitely tender and all contacts, especially with dry surfaces, may produce a paroxysm of pain. Slight jarring and even psychic stimuli are sufficient to precipitate a paroxysm, as in the cases of painful median nerve lesions. The chapter on Causalgia includes a description of these cases (Fig. 269).

Anatomy

The sciatic nerve is the terminal branch of the sacral plexus. It is a large flat ribbon-like nerve about 12 to 14 cm



Fig. 268. Sensory loss in lesions of sciatic nerve following operation.

A. Recovery of pain and cold sense in area of tibial nerve.
B. Return of sensation in tibial, patchy return of pain, and interlacing of cold sense.
C. Marked return of pain sense.
D. Return of pain sense in peroneal; patchy return in tibial; cold sense not recorded.
E. Return of pain sense in tibial; diminished loss of touch sense.

wide and 4 to 5 mm. thick at its origin but it gradually becomes rounder as it descends into the thigh.

It is made up of the ventral and dorsal roots of the fourth and fifth lumbar, first, second and third sacral nerves. The common level of union of these roots is at the anterior border of the great sciatic foramen below the pyriformis muscle. It descends between the greater trochanter of the femur and the

ischial tuberosity through the posterior surface of the thigh to the level of its lower third. There it divides into the tibial and peroneal nerves. However, this division may occur at any



FIG. 269. Macerated skin of foot in case of causalgia of sciatic nerve.

point between the sacral plexus and the popliteal space. As has been shown, the sciatic nerve really consists of two nerves in one sheath: the tibial, which is derived from the anterior divisions and the peroneal, which originates from the posterior divisions.

Just after the nerve appears from the sciatic foramen it lies upon the posterior surface of the ischium, the nerve to the quadratus femoris muscle and the external rotator muscles of

the thigh. It is accompanied by the small sciatic nerve and artery and is covered by the gluteus maximus. In this region it is classically described as lying an equal distance from the great trochanter and the tuberosity of the ischium. In reality it is closer to the ischial tuberosity but its actual position varies with the position of the extremity.

Lower in the thigh and surrounded by its pad of fat it lies upon the dorsal surface of the adductor magnus muscle. It is covered by the long head of the biceps muscle which crosses it obliquely from within externally. The nerve is covered by skin and fascia only at the lower border of the gluteus maximus and at the entrance of the nerve into the popliteal space. Before it divides into its terminal divisions the sciatic gives rise to *articular* and *muscular* branches. Several branches which arise from the upper part of the nerve and may originate from the sacral plexus, perforate the posterior part of its fibrous capsule and supply the hip joint. All of the muscular branches arise from the internal portion of the trunk except the nerve to the short head of the biceps which originates from the external portion of the nerve. The nerve to the *long head* of the *biceps* descends obliquely and externally. It divides at the external border of the muscle into its terminal twigs which ascend and descend to supply the body of the muscle. The nerve to the semitendinosus muscle may arise from a trunk in common with the nerve to the long head of the biceps but otherwise it originates below the latter. A short branch immediately enters the superior part of the muscle while a longer branch enters the inferior portion of the muscle. Both branches lie between the semimembranosus and biceps muscles. The nerves to the *semimembranosus* and *adductor magnus* muscles arise from a common trunk. The small twig to the adductor crosses the semimembranosus and breaks into small twigs as it reaches the inner border of the adductor. The branch to the semimembranosus passes in front of the biceps and semitendinosus and supplies it on its posterior surface. The nerve to the *short head* of the *biceps* descends

parallel to the external border of the sciatic trunk, runs a more or less separate course for 12 to 15 cm. and penetrates the muscle on its medial posterior surface. Stookey states that

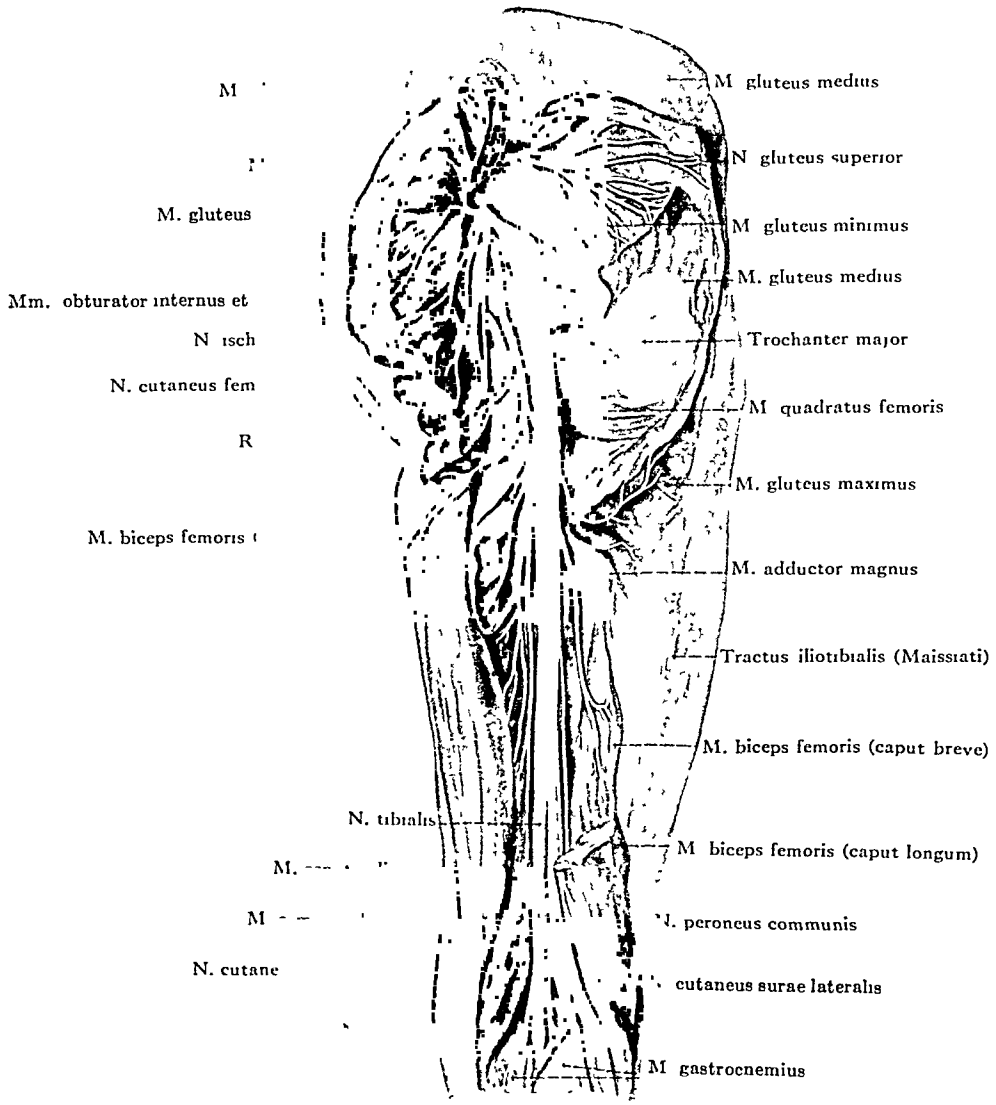


FIG. 270. Anatomical course of sciatic nerve.

the nerve from which the inner hamstring muscles are innervated arises independently from the lumbosacral plexus in

more than 80 per cent of cases and in the remainder it is only incorporated within the sheath of the sciatic trunk. This is of clinical importance, as will be seen later. (Fig. 270.)

Physiology

The *biceps*, *semitendinosus* and *semimembranosus* muscles are flexors of the leg upon the thigh. According to Duchenne they also help extend the thigh upon the pelvis and rotate the leg on the thigh.

Surgery

The sciatic trunk may be pressed upon by various types of pelvic tumors which give rise to pain along its course or which may even produce a complete paralysis. Tumors growing from the pelvic bones, aneurysms of the internal iliac artery or its branches and large bladder calculi may involve the sciatic trunk. Outside the pelvis, violent movements of the hip joint, or dislocations of this joint, may produce a paralytic lesion. It is not uncommon to see patients in whom the nerve has been stretched for the relief of so-called "sciatica" and the resulting intraneural fibrosis may give rise to a lesion which requires surgical interference.

In war, injuries of the sciatic trunk are far more common than any other nerve lesion in the lower extremity. Complete severance of the nerve is not common. For the majority of cases surgery of the sciatic concerns its tibial and peroneal components, to which the clinical symptoms may be confined though scar tissue invòlves the entire trunk. Consequently, one should be familiar with the technique of operative approach to the sciatic and its terminal branches and moreover should be able to apply practically what is known concerning the funicular anatomy of the trunk.

To expose the sciatic nerve in the gluteal region, the patient should be placed upon his abdomen with a sandbag under the opposite shoulder and hip to permit unimparied respirations. The extremity to be operated upon should be flexed slightly

at the knee to relax the hamstring muscles. The limb should be supported beneath the foot by any convenient method so that an assistant's hands are freed. The field should be so draped that, if necessary, the incision may be extended down the thigh.

The incision recommended by Stookey is the most satisfactory. It should begin opposite the posterior inferior iliac spine and be carried downward and outward to a point about three fingers breadth medial to the greater trochanter (Fig. 271). The incision should then curve downward and medially following the curve of the gluteal fold to the middle of the thigh from which point it may be continued downward as far as is necessary. The next step is to undermine the lateral edge of the skin at its inferior margin to expose the attachment of the gluteus maximus muscle. The key to the entire operative exposure is to sever the gluteus maximus at its insertion. This should be done by dividing the upper fibers of the muscle parallel to their course and then severing the muscle attachment about 2 cm. from the femur. In this way the muscle may be reflected medially and upward together with the branches of the inferior gluteal vessels which lie upon its under surface. This avoids the severe hemorrhage and shock of divided muscle fibers which accompanies an incision made directly over the course of the nerve. Such a procedure exposes the nerve immediately from the pyriformis muscle to its entrance into the thigh. After the nerve lesion has been treated, the tendinous attachment of the gluteus maximus should be sutured strongly and little loss of physiologic function of the muscle will follow.

A straight incision which begins over the gluteal fold midway between the ischial tuberosity and the greater trochanter and extends downward in the median line of the thigh will expose the nerve at any point in its course to the popliteal space (Fig. 272). It will be remembered that the sciatic lies more superficially at the popliteal space and just below the border of the gluteus maximus muscle.

In the upper third of the thigh the lower edge of this muscle should be freed in its fascial plane and retracted upward. At the junction of the upper and middle thirds of the thigh

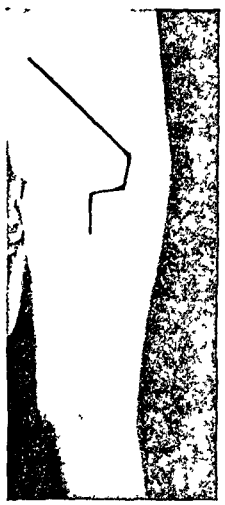


FIG. 271.



FIG. 272.

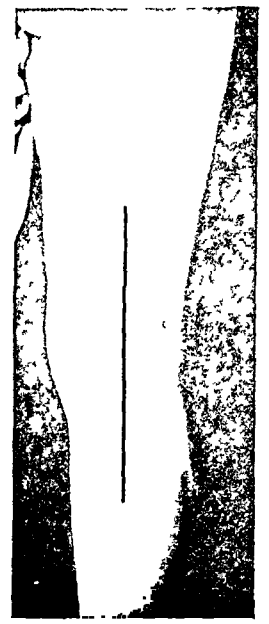


FIG. 273.

FIG. 271. Line of incision to expose sciatic nerve in gluteal region.
FIG. 272. Line of incision to expose sciatic nerve in thigh.
FIG. 273. Line of incision to expose sciatic nerve in popliteal space.

the biceps muscle crosses the nerve obliquely from within outward. Therefore, this muscle should be retracted laterally and the hamstring muscles medially. Occasionally at a slightly higher level it may be easier to retract the biceps medially. The flattened tendon of the semimembranosus muscle may be mistaken for the sciatic trunk which lies immediately along its lateral border surrounded by fatty tissue. In this latter perineural fat will be found the perforating branches of the profunda femoris artery and their accompanying veins.

The sciatic trunk may be so changed in appearance by dense scar tissue that great care should be exercised in identifying the extension of the injury accurately. The tibial division may be involved without injury to the peroneal component or

vice versa. Often the line of demarcation may not be seen and yet can be palpated. It should be remembered that the tibial component lies medially and somewhat anteriorly while the peroneal portion is located on the posterolateral aspect of the nerve. Consequently, the line of cleavage runs somewhat obliquely. It may be necessary to identify the line of cleavage nearer the popliteal space where the two nerves separate to run independently and then follow this plane upward. The use of the stimulating electrode will help in this identification, besides it furnishes evidence of the presence or absence of fiber conductivity.

A linear incision should be placed in the medial line of the popliteal space to expose both of the divisions of the sciatic nerve (Fig. 273). Both nerves lie rather superficially beneath the popliteal fascia, fat and the lower portions of the hamstring muscles. The popliteal artery and vein lie in a deeper plane. The external saphenous vein enters the popiteal vein and may be obliterated by the presence of a large amount of scar tissue. It is wise to ligate the vein so that it may not be torn off and serious bleeding from the popliteal vein is thus avoided. The latter structure is commonly involved in scar tissue and its walls are easily torn so that it must be handled with care. Attempts to remove the bed of scar tissue may have to be abandoned because of possible injury to this vein. The tibial nerve descends through the middle of the space and passes beneath the superficial muscles of the back of the leg. The peroneal nerve descends obliquely, crosses the lateral head of the gastrocnemius and passes superficially to the head of the fibula. If the dissection is started from above downward it will be easier to identify the muscular branches of the tibial which innervate the two heads of the gastrocnemius muscle. The cutaneous nerves which arise in the popliteal space need not be repaired if their union is a difficult surgical procedure because their supply is overlapped by the internal saphenous and musculocutaneous nerves. However, they should be identified so that they will not be sutured to the motor branches.

Chapter XXXI

THE PERONEAL NERVE

Somewhat analogous to the radial nerve, when injured a lesion of the peroneal nerve produces immediate and extensive paralysis of all the muscles supplied by it. As a result of the injury, a deformity characterized by foot-drop is observed. In addition to the foot-drop, a slight drop of the first phalanx of all of the toes may be seen. At times a dorsal tumor of the tarsus, which is usually due to the more or less pronounced projection of the astragalus and scaphoid bones and to the thickening of the sheaths of the extensor tendons is observed (Fig. 274).

Motor Symptoms

When examining voluntary motion it is necessary to immobilize the knee of the patient, and to be alert unless twitchings of the aponeurosis of the leg caused by contraction of the muscles of the thigh be mistaken for contraction of muscles supplied by the peroneal nerve. Under careful observation, no active movement of dorsal flexion of the foot is possible. Adduction of the foot is executed by the tibialis posticus. Extension or dorsal flexion of the proximal phalanges of the toes is impossible. The distal phalanges may be extended by the contraction of the interosseus tendon, which in the foot as in the hand extends the last two phalanges. Abduction of the foot cannot be performed (Fig. 275).

A steppage gait is characteristic. The patient walks by raising the knee high, the point of the foot is dropped and adducted. It is planted heavily upon the ground. The disability is commonly greater than when the entire sciatic nerve is severed.

Supplementary Movements: Supplementary movements of the ankle joint are observed rather frequently. Strong flexion

of the toes occasionally results in inversion and slight dorsal flexion of the foot, due to a mechanism similar to that observed in which strong flexion of the fingers produces passive exten-



FIG. 274. Characteristic deformity in peroneal nerve paralysis.

sion of the hand in radial nerve lesions. Movements of the toes are sometimes confusing when the contraction of the antagonists of the paralyzed muscles is followed by a rebound simulating the normal action of the muscles under investigation. If the dorsal flexors of the toes are paralyzed and the

patient attempts to contract the paralyzed muscle, plantar flexion of the toes may be the initial movement, followed by a rebound of the toes to the original position.

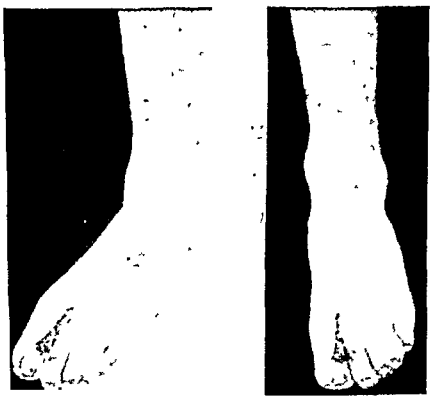


FIG. 275. Inability to abduct foot in peroneal nerve paralysis.

SENSORY SYMPTOMS

Subjective sensory disturbances are rare. When present, they consist of aching pains in the upper outer part of the leg. At times the patient complains of a deep-seated pain in the external malleolus. When pain has been a prominent factor, the injury has been above the bifurcation of the sciatic and a slight injury to the tibial nerve has existed at the same time.

Objective sensibility is destroyed over the dorsal surface of the foot and the anterior and outer surface of the leg. In the foot, sensation is lost to the base of the toes, extending outward to a point between the fourth and fifth toes, and inward to the base of the first metatarsal bone. The upper limit varies, but in many instances may extend to the head of the fibula (Figs. 276 and 277). The borders adjacent to uninjured nerves are sensitive to pin prick and to the extremes of temperature. The isolated supply of the peroneal nerve is often surprisingly small, as was found to be the case in radial nerve lesions.

The area of exclusive supply of pain of the peroneal nerve was obtained from certified cases of division and cases which

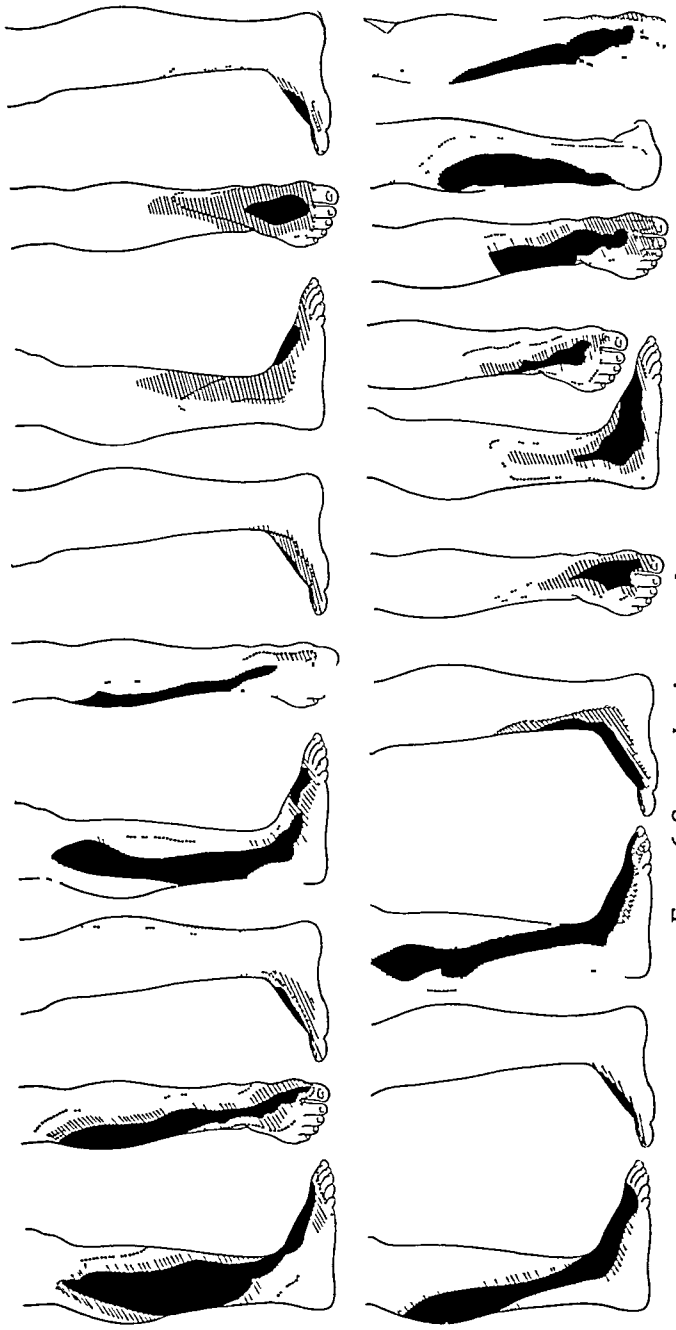


FIG. 276. Sensory loss in peroneal nerve lesions.

were examined less than thirty-seven days following resection and suture. The area consists of a narrow band which extends from a point a little above the junction of the lower and middle

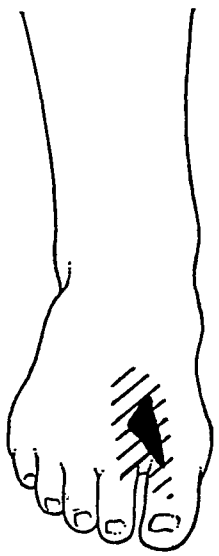


FIG. 277. Sensory loss in partial lesion of peroneal nerve with superficial peroneal nerve undivided.

thirds of the outer surface of the leg, diagonally across the dorsum of the foot to a point over the middle of the metatarsal bone of the great toe. It is interrupted at the junction of its lower and middle thirds by an area which is sensitive to pin prick. The area is due to the overlap on one side of the long saphenous nerve and on the other side of the tibial nerve (Fig. 278). Although a number of cases showing such an interruption in the band of analgesia have been observed, they have not fulfilled the requirements which we demanded in estimating exclusive supply. One case showed this type of interruption of the band of analgesia twenty-seven days after resection and suture.

Our results, therefore, are at variance with those of Head, Rivers and Sherren. Referring to the fact that the unit for protopathic sense is the posterior root, they said that following

section, the peroneal nerve, which represents a large part of the fifth lumbar root, does not show a great difference between borders of the areas of analgesia and anesthesia.

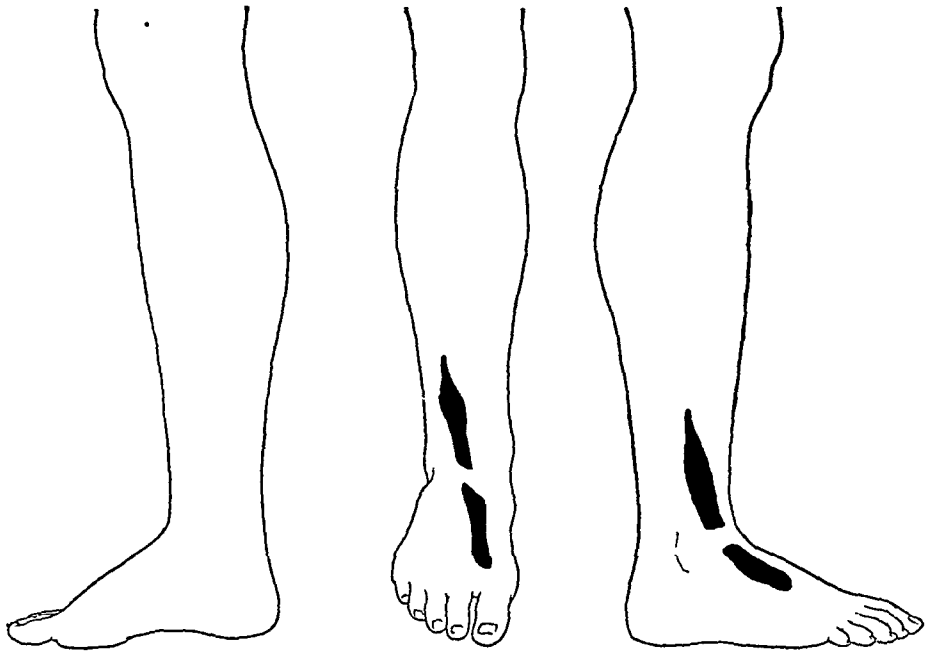


FIG. 278. Isolated supply of peroneal nerve to pain.

PARTIAL LESIONS

Cases of incomplete and dissociated paralysis of the peroneal are observed, but not frequently. They generally occur following wounds in the leg in which the superficial or the deep peroneal nerve has been injured alone. Dissociated paralysis may occur, however, in lesions above the bifurcation of the nerve. Under this condition we may have a paralysis of the tibialis anticus alone, or of the peronei muscles alone, while the extensors of the toes are spared. Occasionally the extensors of the toes may be paralyzed and the other muscles may be only slightly involved or completely spared.

Signs of Motor Recovery

The order of the recovery of motion is given by Bénisty as tibialis anticus, peronei, extensor longus digitorum and exten-

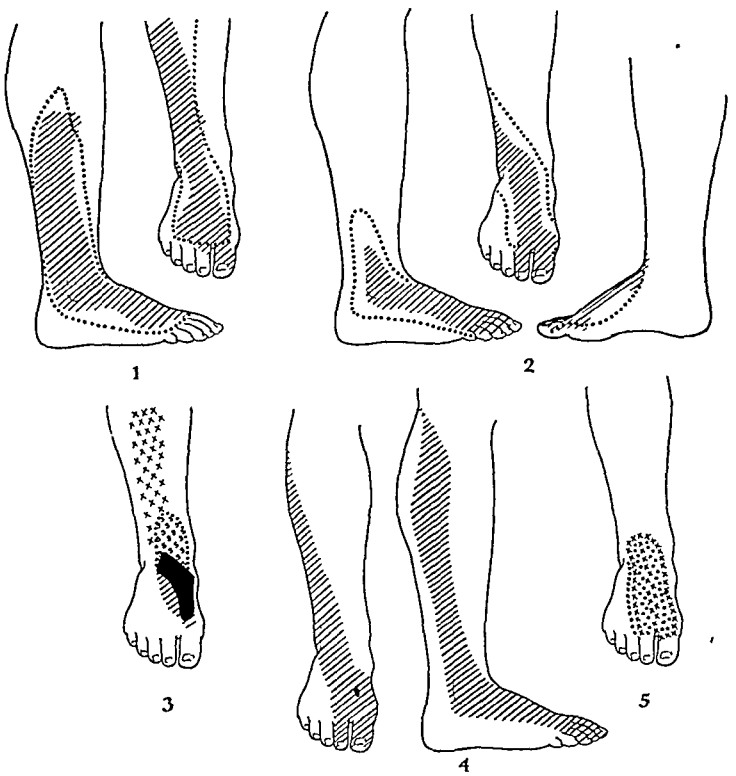


Fig. 279. A. Sensory loss in peroneal nerve lesions which recovered spontaneously. 1. Recovery of pain sense; interlacing of cold. 2. Recovery of pain sense. 3. Recovery of touch and cold sense, not pain. 4. Diminished area of loss of pain sense. 5. Recovery of touch, not pain sense.

sor proprius hallucis. Stopford agrees with this, whereas the records of the Military Orthopedic Hospital place the extensor longus digitorum first. In a small group of 29 cases in the American material which recovered spontaneously, the tibialis anticus recovered in 22 cases; the peronei in 7; the extensor longus digitorum in 3; and the other muscles in 3. Of 27 cases which had peroneal nerve sutures, 6 showed recovery. The order of return of motion was in the tibialis anticus, extensor longus digitorum and the peronei.

Signs of Sensory Recovery

Peroneal nerve lesions which recovered spontaneously frequently showed complete recovery to pain. Interlacing of the

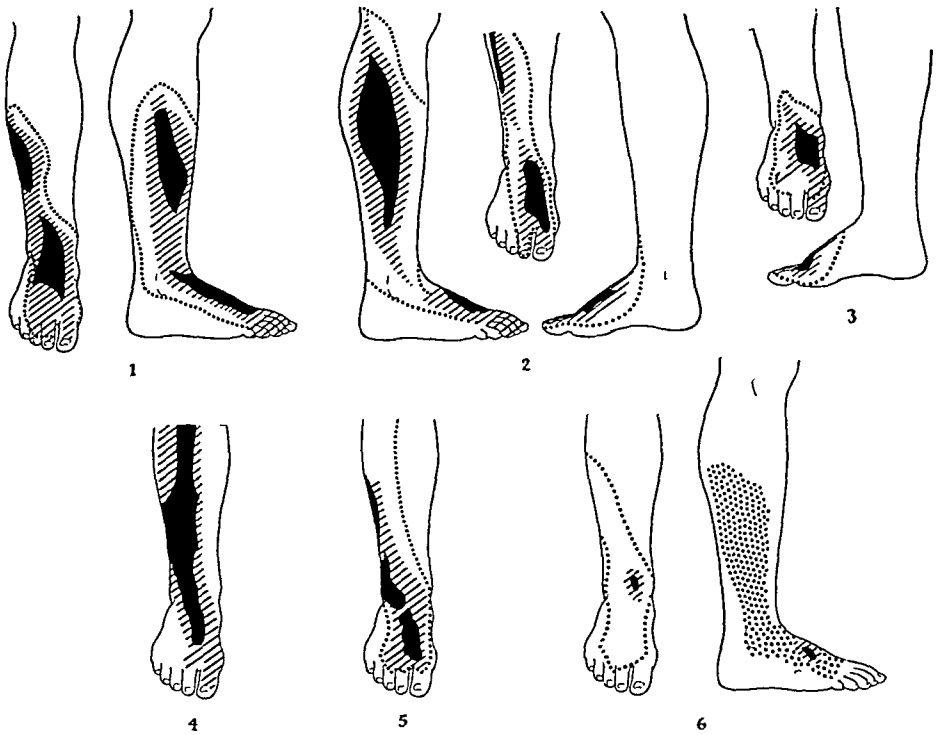


Fig. 279. B. Sensory loss in peroneal nerve lesions after operation. 1. Patchy return of pain sense. 2. Patchy return of pain and touch sense; primary suture. 3. Return of touch sense in area of isolated supply. 4. Recovery of cold sense, great toe; interlacing. 5. Patchy return of pain sense; interlacing. 6. Recovery of touch sense, not cold.

border of loss of cold was seen, particularly over the dorsum of the foot. Areas of recovery to touch and not to pain were occasionally observed. In the patients who recovered following operation, loss of pain sense was more common. In those cases patchy return of pain sense was observed very frequently. Interlacing areas which showed recovery of touch and not of cold, as was the case in the radial nerve, were noted (Fig. 279).

Anatomy

The peroneal nerve is about one-half the size of the other terminal branch of the sciatic trunk. As was pointed out it arises from the posterior divisions of the last two lumbar and the first two sacral nerves.

After its origin from the bifurcation of the sciatic trunk it descends obliquely across the popliteal space close to the medial margin of the biceps muscle to the head of the fibula. It passes between the biceps tendon and external head of the gastrocnemius muscle and winds around the neck of the fibula. It divides into the *deep peroneal* (anterior tibial) and *superficial peroneal* (musculocutaneous) nerves between the peroneus longus muscle and the bone (Fig. 280).

Previous to the division into its terminal branches the nerve gives off articular and cutaneous branches. Two of the articular branches accompany the superior and inferior external articular arteries to the outer side of the knee. The third branch arises at the point of division of the peroneal nerve. It accompanies the anterior recurrent tibial artery through the tibialis anticus muscle to supply the anterior surface of the knee joint. There are usually three or more cutaneous branches which supply the skin along the posterior and external surface of the leg. The largest branch (communicans peronei) arises near the head of the fibula, crosses the external head of the gastrocnemius to the middle of the leg and joins with the communicans tibialis to form the *external saphenous* nerve.

The *deep peroneal* descends obliquely beneath the extensor longus digitorum muscle to the anterior part of the interosseous membrane. It then descends with the anterior tibial artery to the anterior surface of the ankle joint. Here it divides into an *external* and an *internal* branch. The relation of the nerve to the artery is interesting in that it first lies lateral, then anterior and finally lateral to the artery at the ankle joint. The deep peroneal nerve in its course through the leg innervates the tibialis anticus, extensor longus digitorum, peroneus tertius

and extensor proprius hallucis muscles. It also supplies an articular branch to the ankle joint. The *external* or *tarsal* branch of this nerve passes laterally across the tarsus beneath

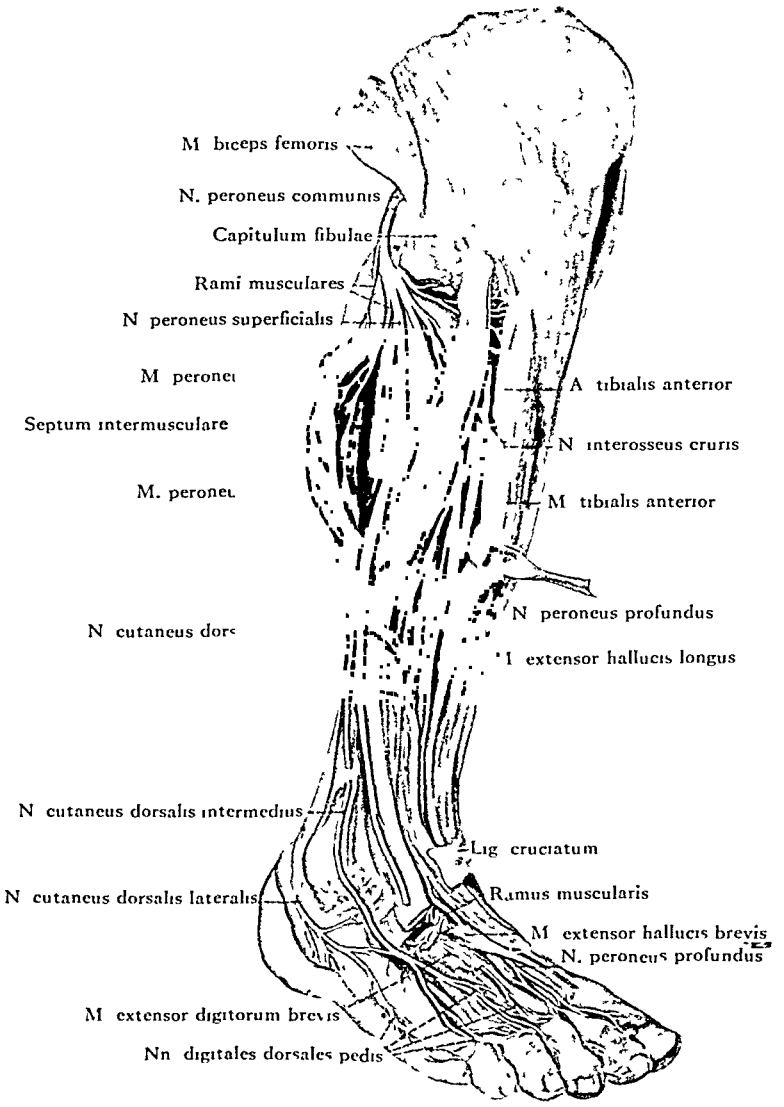


FIG. 280. Anatomical course of peroneal nerve.

the extensor brevis digitorum which it supplies. It becomes enlarged slightly and gives off three small *interosseous* branches which supply the tarsal joints, the metatarsophalangeal joints

of the second, third and fourth toes and the second dorsal interosseous muscle. The internal branch is in reality a continuation of the parent trunk. It accompanies the dorsalis pedis artery along the inner aspect of the dorsal surface of the foot. It divides into two dorsal digital branches which supply the adjacent sides of the large and second toes. It also gives a twig to the metatarsophalangeal joint of the great toe and a branch to the first dorsal interosseous muscle.

The superficial peroneal passes anteriorly between the peronei muscles and the extensor longus digitorum. It pierces the deep fascia at the lower third of the leg on its anterior and lateral side. Before dividing into two cutaneous branches it supplies the peroneus longus and brevis muscle. The *internal dorsal cutaneous* branch passes anteriorly to the ankle joint and divides into three dorsal digital branches. The internal of these supplies the skin of the dorsum of the foot and the inner side of the great toe. The middle branch supplies the adjacent surfaces of the great and second toes. The external branch supplies the adjacent sides of the second and third toes. The *external dorsal cutaneous* branch passes along the external surface of the dorsum of the foot and supplies the adjacent sides of the third and fourth and fourth and fifth toes.

Physiology

The *tibialis anticus* muscle flexes the foot dorsally. It also aids in adduction and rotation of the foot.

The *extensor longus digitorum* extends the proximal phalanges of the four smaller toes. It aids in dorsal flexion and abduction of the foot. The *peroneus tertius* acts in conjunction with this muscle.

The *extensor proprius hallucis* extends the first phalanx of the great toe.

The *peroneus longus* muscle produces plantar flexion of the foot, lowers the inner side of the ball of the foot and turns the foot externally as the lateral border rises. This imparts a movement of abduction and rotation to the foot.

The *peroneus brevis* abducts and everts the foot.

The *extensor brevis digitorum* aids in extension of the

proximal phalanges of the first four toes.

Surgery

To expose the peroneal nerve the patient should be placed

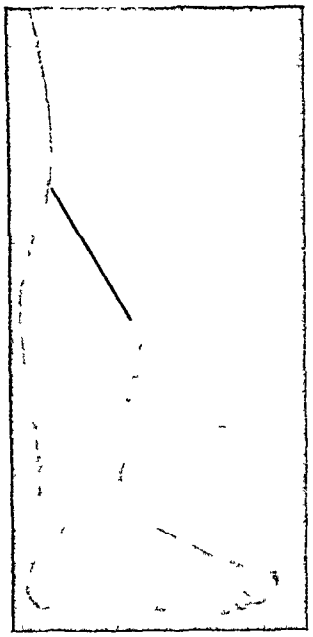


Fig. 281. Line of incision to expose peroneal nerve.

in a semi-prone position and the leg should be slightly flexed and held in internal rotation. The incision should extend obliquely from the middle of the popliteal space along the medial border of the biceps tendon and across the fibula about an inch below its head (Fig. 281). The poplietal fascia is divided by a parallel incision to that in the skin. The nerve may then be identified beneath the edge of the biceps tendon. The nerve becomes flattened somewhat as it crosses the fibula. It is covered by a thick layer of connective tissue. Two definite fiber bundles may be seen in the peroneal as it leaves the popliteal space. Those fibers which form the anterior tibial nerve lie adjacent to the biceps tendon and consist of one or two funiculi; fibers which make up the musculocutaneous lie more lateral and dorsal and consist of three funiculi. A small artery and vein occasionally lie between these bundles.

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LIGHT FROM THE LABORATORY AND THE CLINIC ON THE CAUSES OF PEPTIC ULCER*

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FOR more than fifty years able men have been struggling with the problem of peptic ulcer, trying to find out why the stomach does not ordinarily digest itself, why acute ulcers heal, and why chronic ones either fail to heal or else, when they do heal, tend to become active again.

ACUTE EXPERIMENTAL ULCERS

I shall not attempt to review here the enormous literature that has grown up about the subject of ulcer causation; von Redwitz and Fuss did not waste any words and yet their splendid analysis of the subject covers 387 pages. Hauser's great monograph, which represents notes collected during the course of a long life, is even more extensive. All I can hope to do here is to comment on a few of the discoveries that seem to me to throw light on the problem of chronic peptic ulcer as it appears to the clinician, the surgeon and the physiologist.

As Cohnheim saw so clearly in 1880, the difficulty facing the experimenter has not been to produce ulcers; his trouble has been to make chronic ones that will not promptly heal. Many laboratory workers in the last fifty years have snipped out bits of mucosa from the stomachs of animals; they have injected caustics and toxins and bacteria and have produced necrotic areas or hemorrhagic patches, but except in rabbits (Ferguson, Fauley and Ivy), these defects have always healed over promptly just as do the wounds made by surgeons in the stomach and duodenum of man.

THEORIES OF ULCER CAUSATION INADEQUATE

There are a number of theories of ulcer causation, some helpful but none entirely satisfactory to the clinician because they do not explain the peculiarities of the disease as it occurs in man. Thus they do not tell us why in this country men are more subject to the disease than women, and why in some other parts of the world men and women are equally affected. They do not explain why jejunal ulcer is seen ten times as often in men as in women, and they do not help us to understand why today, in America, duodenal ulcer is so much more common than gastric ulcer when in some parts of Europe the reverse appears to be true now or to have been true in the past. They do not explain why duodenal ulcer tends to come in the twenties and thirties and why gastric ulcer tends to come in the thirties and forties. They do not explain why gastric ulcers usually cease growing when they are 2.5 cm. in diameter and why duodenal ulcers are only half as large. One wonders why

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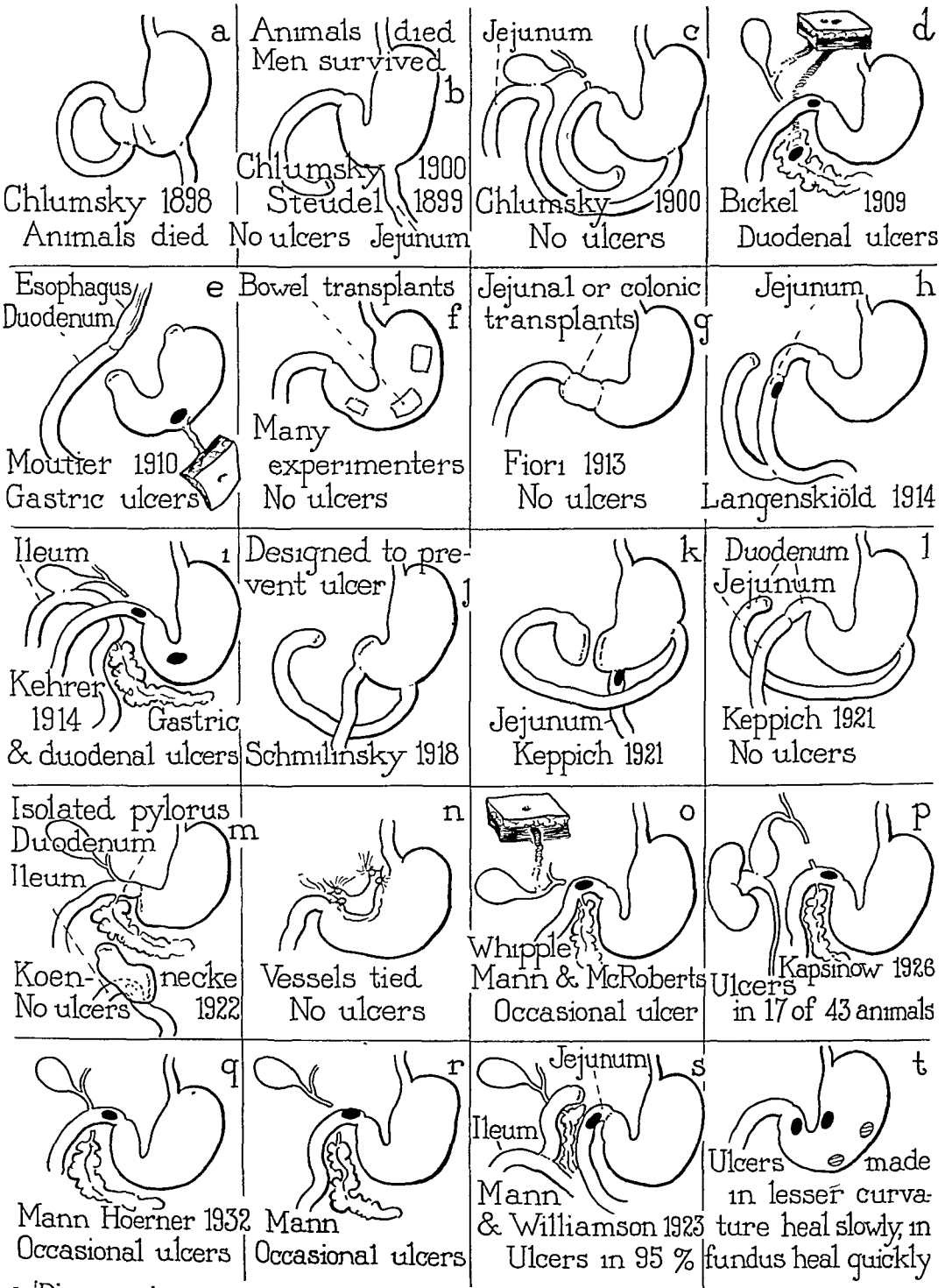


FIG. 1. Diagrammatic representations of operations performed on dogs with the hope of throwing light on the causes of peptic ulcer.

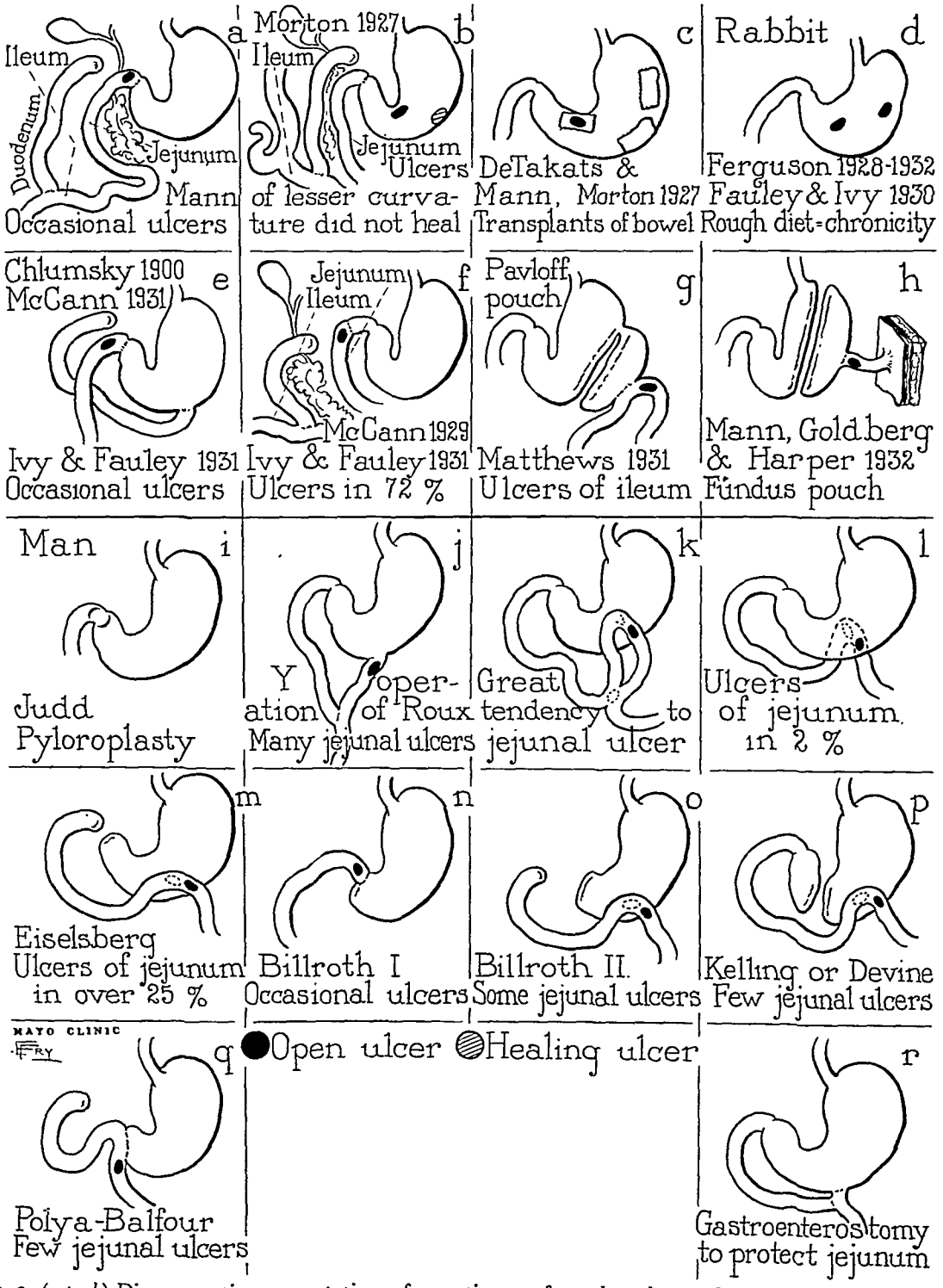


FIG. 2. (*a* to *b*) Diagrammatic representations of operations performed on dogs and rabbits with hope of throwing light on causes of peptic ulcer. (*i* to *q*) Diagrammatic representations of operations performed on man with the hope of curing ulcer. (*r*) A suggested operation to cure ulcer.

there should be any limit to the destructive process once it is started. Why also are ulcers so often single? It would seem as if the causes that produce one might just as well produce another. Furthermore there is no entirely satisfying explanation of the fact that perhaps nineteen out of twenty ulcers are to be found in either one of two small areas in stomach and bowel. Every so often a writer thinks he has answered some of the questions about ulcer only to realize later that while his explanation may be applicable to lesions in the duodenum it is useless when it comes to explaining those in stomach and jejunum.

ARTERIAL CHANGES

A number of investigators have shown that ulcers can form in the stomach of man when one of the gastric blood vessels is narrowed or plugged, and some have suggested that the disease is commonly due to arteriosclerosis or embolism. End-arteritis can doubtless be demonstrated in the neighborhood of some ulcers, and, when present, it should have a restraining influence on healing. In many cases of ulcer, however, it has been impossible to show much if anything wrong with the vessels, either in stomach or duodenum. In other cases the arterial changes have been found only in or around the floor of the ulcer, in which case the natural supposition is that they are secondary to inflammation, and that they serve a useful purpose in protecting the patient from hemorrhage.

If arterial degeneration were a common cause of ulcer, one would expect to find the disease beginning commonly in elderly people and not, as is the case, in youths and young adults. It may well be, however, that arteriosclerosis is the cause of some ulcers, and particularly of those which form late in life.

Although actual arterial *disease* does not appear to be a common cause of ulcer, especially in young people, the possibility remains that arterial *spasm* is one of the links between the emotions and ulcer. An argument against these theories is to be found in the fact that a number of investigators have been able to tie off from one-third to four-fifths of the gastric blood supply in animals without producing necrosis (Fig. 1*n*). In some cases they could show, with the help of vital stains, that an injury had been wrought to the cells in the affected area of mucous membrane, and in a few instances acute ulcers were produced. It seems to me probable that when certain vessels are tied, blood continues to reach the mucous membrane by way of connecting branches, and injury does not result, but if a vessel is closed throughout its length by spasm or disease the cells dependent on it for blood must die. Thus, when Payr tied off large vessels to the stomach he occasionally produced an acute ulcer but when he injected into a small gastric artery a few drops of a dilute solution of formol he obtained, in a number of the animals, chronic and even perforating ulcers. Subsequent examination showed that the lumen of the injected vessels had been much narrowed.

Many experimenters reported also that they could produce superficial ulcers by poisoning animals with sublethal injections of pilocarpin and physotigmin. The drug produced so much spasm of some of the gastric vessels that small round areas of anemic or cyanotic muscle and mucosa could be seen to form. According to Gallagher acute ulcers formed in the duodenum whenever he clamped the mucous membrane and made it anemic for more than thirty minutes. We have here, then, a possible explanation for the small size of most ulcers: that is, their area corresponds perhaps to that supplied by a small artery.

The question next arises: But why doesn't an erosion made in this way heal as soon as the spasm relaxes? Certain experimental work suggests that the presence of a defect in the mucosa tends to draw to it or concentrate about it all the tendency to muscular spasm that is engendered in stomach and duodenum by

an overly irritable vagus nerve, and it may be that in this way a vicious circle is set up. There is some experimental evidence to show also that harmful substances can pass through the ulcer and into the sheath of the vagus nerve to cause even more irritability.

Against the theory of ulcer causation through arteriolar spasm is the observation of Stahnke that long-continued electrical stimulation of the vagus nerves caused relaxation of the gastric vessels at the same time that it produced hyperperistalsis and powerful contraction of the muscle. Stahnke doubted if Nature would have made a nervous mechanism that would increase muscular and secretory activity and at the same time shut off the supply of much needed blood.

Reeves, Disse, Jatrou, Hoffman and Nather, Berlet, and others have shown that the small blood vessels supplying the mucous membrane in the ulcer-bearing zone along the lesser curvature of the stomach and in the duodenal cap are peculiar and perhaps more like endarteries than are the vessels elsewhere in the digestive tract. Working independently, they all came to the same conclusion: that the duodenal cap is poorly supplied with blood and therefore markedly predisposed to injury and to poor healing afterward.

Histologists have pointed out also that the mucous membrane along the *Magenstrasse* is much like that in the intestine. Besides, it is so firmly attached to the muscle that it cannot slip away from foreign bodies that may impinge upon it. It may be significant that the mucous membrane in the duodenal cap is also firmly attached to the muscle. Bauer has emphasized the point that the lesser curvature of the human stomach is, in a way, a vestigial organ in that it represents what is left of the furrow which in some of the lower animals carries liquids into the pyloric stomach. Like some other vestigial structures it may be particularly subject to disease. Against this idea is the probability that man comes of a stock that never possessed a complicated stomach of the ruminant type.

One is tempted then, to look to definite histologic peculiarities for an explanation of the fact that in man the region in the stomach near the incisura is so highly susceptible to ulcer until one learns of the work of de Takats and Mann, Morton and others who showed that segments of intestinal mucosa transplanted into the wall of the stomach seldom become ulcerated except when they are placed along the lesser curvature (Fig. 2 *c*.). These experiments show that the susceptibility of this region cannot be ascribed wholly to anatomic peculiarities but must be due at least partly to its exposure to some extra strain or mechanical or chemical injury. In conformity with this view is the fact established by many investigators, that acute ulcers heal faster when they are made in the fundus of the stomach and along the greater curvature than when they are made in the *Magenstrasse* (Clairmont, Morton, and others).

As is well known, there is almost no peristalsis in the fundus and little chance for mechanical injury to the mucosa. Against this theory of mechanical stress however is the fact that if friction were the most important factor in determining the location of ulcers in the stomach, then they should be found commonly in all parts of the pars pylorica and particularly in the pyloric ring. Actually they seldom occur there. One explanation offered is that the attachment of the mucous membrane to the muscle in the pyloric region is so loose that the mucous membrane can slip away from an impinging body such as a piece of bone.

Many physicians have assumed that in man the *Magenstrasse* or gastric furrow carries fluids from cardia to pylorus much as it does in some of the lower animals. My own observations, together with those of several roentgenologists, make me doubt if this is true. When, on a few occasions, I gave barium and water to a person who had just dined, the fluid seemed to run

over the mass of food in the stomach on all sides much as frosting would run over a cake, and I could not see that any large amount followed the lesser curvature. (For the literature see Hauser, p. 698.)

INFECTIOUS THEORY

Since the work of Böttcher in 1874, the literature has been full of reports of cases that show that at times infection can give rise to acute ulcers or, more often, to transient hemorrhagic erosions. The infectious theory of ulcer causation leaves much to be desired, however, because it does not throw light on any of the peculiarities of the incidence of the disease. Thus, it does not seem likely that focal infection is three times as common in men as in women, and I doubt if it is much more common at the age of twenty-five than it is at forty-five years. Besides, time and again I have seen ulcer symptoms return to disable a patient whose discoverable focal infections had all been removed, and just as frequently I have seen an ulcer heal spontaneously and stay healed for a long time in a patient whose mouth was full of devitalized teeth and carious snags.

The work of Rosenow, Haden and Bohan, Nickel and Hufford, and others is suggestive but as yet it is hard to say what relation the hemorrhagic patches and acute ulcers produced by them in rabbits have to the chronic ulcers of man. Incidentally, Ivy has produced hemorrhagic patches by injecting sterile broth cultures containing lampblack which makes one wonder if the bacteria do not at times serve as simple emboli in the small gastric and duodenal vessels. On the other hand several experimenters have obtained the same type of lesion by injecting bacterial toxins alone. According to Emery and Monroe foci of infection are no more common in patients with ulcer than in those without it. Unfortunately the difficulties to be overcome in gathering sufficient data to prove or disprove the value of removing focal infections in cases of chronic ulcer are so great that I doubt if the task can ever be accomplished to the satisfaction of everyone. In the meantime the physician may remove focal infections on general principles or because he has faith that such work will cure ulcer, but I do not see how he can promise the individual patient anything.

I like to clean up those lesions in mouth and nose which can produce a steady stream of pus down the esophagus. I have no statistical proof with which to back up my belief but it seems to me logical to assume that an ulcer should heal better when not bathed day and night in infectious material. Against this idea is the fact that ulcers are seen most commonly in the men and women who have the most bactericidal gastric juice.

One of the difficulties in analyzing the influence of infection on ulcer arises in the fact that in so many cases foci immensely larger and more important than those in teeth, tonsils, or prostate gland are present and unrecognized in appendix, gallbladder, liver, mesenteric lymph nodes, and, in the case of perforated ulcers, in the mass of inflammatory tissue surrounding the lesion.

The most fertile source of infection producing flare-ups of ulcer symptoms seems to be the common cold. As Eusterman has pointed out, gastric hemorrhages often follow an attack of influenza or sinusitis. Occasionally the infection appears to enter through the skin; thus, I know of two physicians who suddenly found themselves with acute and very painful duodenal ulcers, demonstrable with the roentgen ray. One had a felon on the finger and the other an infected wound. These ulcers healed symptomatically and the crater disappeared a few days after the source of infection was removed. Most of us probably suffer from time to time with acute ulcers of infectious or nervous origin just as we suffer with canker sores or herpes labialis. The fortunate thing is that in those of us who are perhaps free from a "spasmophilic tendency" or an "ulcer diathesis," these lesions promptly heal.

TYPE OF PATIENT WHO GETS ULCER

Before launching into a study of the methods used for the production of experimental ulcer it might be well to review briefly a few of the clinical peculiarities of the disease. There is no question that it tends to run in families, and that the hereditary factor is at times important. What we all need to know now is the nature of the inherited defect or deficiency. Much evidence is accumulating to show that in many cases the tendency to the formation of ulcers is based on the abnormally high reactivity of the individual. He is often a keen, wide-awake, sensitive type of man who drives himself all day and part of the night, who responds too much to things that break into his routine, and who does not know how to relax.

Anyone with large clinical experience will doubtless feel inclined to remind me that many patients with ulcer do not conform to this type, and that many persons of this type never suffer with ulcer. While admitting this, I must point out that many persons who are outwardly calm are inwardly tense, anxious, fearful, or actually seething. To me it is a curious fact that in many persons tenseness and nervousness do not seem to give rise to any disease while in others they tend to produce perhaps a nervous breakdown, attacks of migrain or mucous colitis, or a steadily mounting blood pressure. Other nervous persons suffer for years with an ulcer-like syndrome, the mechanism of which is not yet known.

PSEUDO-ULCER

A few of the patients with pseudo-ulcer eventually are found to have one or more ulcers, but most of them live on for years without the development of a definite lesion. Often the symptoms are so striking that the clinician must wonder if the negative report from the roentgenologist is correct, but we at The Mayo Clinic have found from long experience that especially when the history is somewhat atypical, when there is distress before breakfast and none at night, and when the symptoms have been practically constant for years without the customary remissions, and without the appearance of signs of obstruction or perforation or hemorrhage, it seldom pays to disregard the evidence obtained in the dark room. Too often at operation on such patients the most careful search will fail to reveal an ulcer.

Some of the patients with pseudo-ulcer or with ulcers that tend to recur and become disabling in spite of every form of medical and surgical treatment have at all times a marked and uncontrollable hyperacidity, but others do not, and not infrequently one sees a person whose ulcer refuses to heal in spite of the fact that the acidity is so low that there cannot be much peptic activity in the stomach. Furthermore there are persons who live out their lives with a gastric acidity far above normal and yet never show symptoms or signs of ulcer.

HYPERACIDITY IS NOT THE ONLY CAUSE OF ULCER

In mentioning the presence of low acids in some cases I do not wish to obscure the fact that a persistently high acidity is often a prominent characteristic in cases of severe and intractable ulcer; I only wish to warn the reader against a too ready acceptance of the popular view that hyperacidity is the sole factor in the production of the disease. Recent studies (Vanzant et al.) show that gastric ulcers are associated, on the average, with acidity slightly lower than normal, and one has only to see one of the rare cases of chronic ulcer and no free gastric acidity (by ordinary tests) or a few cases of elderly men with a free acidity around 80 units and no sign of ulcer, to realize that other factors besides acidity must often enter in before an ulcer can be formed or maintained in a chronic state.

Mann and Bollman have had every reason to be impressed by the importance of acidity in the production of experimental

ulcers, and they and their students have shown clearly enough that the duodenum or jejunum that succumbs to ulceration probably does so because it is exposed too long to concentrations of acid which are higher than normal for the particular region involved. This factor of unusual or prolonged exposure impresses me more than the one of simple gastric hyperacidity. When I remember how large the factors of safety are in most of the functions of the body it is hard for me to believe that an increase in gastric acidity to only one and a half or at most to twice normal could, alone, be responsible for the formation of ulcer. I know it is possible but I have reasons for suspecting that future research on patients with ulcer will show an absence from the gastric juice of some substance which normally tends to protect the mucous membrane from injury by hydrochloric acid. (See Matthes, Ivy and Fauley, Mann and Bollman.)

Curiously, Dragstedt found years ago that acute ulcers produced in a gastric pouch made so as to retain its secretion, healed as quickly as did ulcers in a pouch in which the juice was not retained. Somewhat against this observation is that of Hughson, Bolton, Friedman and Hamburger, Ivy and Fauley and others, who found that the production of some degree of obstruction at the pylorus, which usually increases the acidity, delayed the healing of experimentally produced gastric ulcers. By itself, experimentally produced pyloric stenosis rarely seems to lead to the production of ulcers. Slocumb is the only one whom I have found reporting much success with this technique.

DIFFERENT TYPES OF ULCER IN MAN

An important point to be kept in mind is that there are doubtless *several causes for ulcer* and several types of the disease in different parts of the world. This was brought home forcibly to us at The Mayo Clinic by Walters and Snell who recently went to Germany to see why the gastroenterologists in that country are always talking about a gastritis that precedes or is associated with ulcer. We were puzzled about it because we so rarely see it in Rochester. Actually Walters and Snell found that in some parts of Germany duodenal ulcer is usually associated with a diffuse inflammation and ulceration of the mucous membrane of the distal end of the stomach, and they could easily understand why, under the circumstances, European surgeons like to resect widely. Later, Dr. Sebening of Frankfurt spent some time with us at the Clinic and was equally impressed by the fact that the pathology of ulcer is different in the United States and in Germany. Recently Aschner showed that many of the bits of stomach resected in New York contain multiple lesions resembling those seen in Germany. The next question is: Why are these differences so marked?

Perhaps as time goes on, much can be learned about the causes of ulcer by studying its incidence in different countries and among peoples who have different dietary customs. I need but mention the reported high incidence of ulcer in Abyssinia where the people take their food covered with pepper (Bergsma), or the peculiar distribution of ulcer cases in Southern India where a diet of tapioca is supposed to be responsible for the disease (Sommervell, McCarrison). It appears to be unusually common also in Denmark and among soldiers stationed in Hawaii.

It has interested me much to find in Minnesota a marked tendency of ulcer symptoms to flare up in the spring and in the fall. I do not remember having seen this in California where the seasons are not so marked, and Larsen of Honolulu writes me that it is not seen in that winterless climate.

WHY DOESN'T THE STOMACH DIGEST ITSELF?

The earliest workers on the problem of ulcer were naturally concerned with the question: why doesn't the stomach digest

itself? At first its immunity was thought to be due to the alkalinity of the circulating blood and later to a postulated antipepsin. When an antipepsin could not be demonstrated to the satisfaction of everyone, most experimenters fell back on the statement that there is something about the *living* cell that protects it. They transplanted the spleen and kidney and bits of bowel into the wall of the stomach, and so long as the circulation of the graft was unimpaired there rarely was any sign of digestion in it (Fig. 1 *f*). (Hotz, Exalto, de Takats and Mann, Morton, Dogliotti and Riccio, and Dragstedt and Vaughn.)

Necheles, Ling and Fernando found that healthy living tissues implanted into the duodenum or upper jejunum were not digested. When, however, they injured the pancreas by injecting sterile olive oil into the main duct, digestion of the implants took place and the mucous membrane of the whole stomach and small intestine showed signs of hemorrhagic inflammation.

According to Michaelis the living cell is immune to peptic activity probably because its limiting membrane contains lipoids which cannot be attacked by a protein-splitting ferment. This theory sounds plausible until one gets to wondering why activated pancreatic lipase does not initiate destruction of the duodenal mucosa and why cells are not digested by their own proteinases.

Actually, in many instances, the first and all-essential injury to the limiting membranes of the gastric or duodenal cells is probably wrought by strong concentrations of hydrochloric acid or by gastric juice which is not modified in some way by duodenal secretions. The work of Matthes, reported in 1893, shows that the mucous membrane of the living bowel is promptly injured by hydrochloric acid of from 0.2 to 0.5 per cent concentration. He used dogs with Thiry fistulas. Strange to say, natural gastric juice from a pig was much less irritating than artificial juice made by adding pepsin to dilute hydrochloric acid. He tried adding mucin to the artificial juice to see if he could temper its action in this way but without result.

Langenskiöld repeated Matthes' work, using large amounts of pure gastric juice from dogs. This juice was run into the stomach through a catheter and out through another catheter placed in the duodenum. The animals were then killed and the mucous membrane studied. Especially when the juice had been in contact with the mucous membrane for as long as three hours excoriations were found and sometimes ulcerations which extended down to the muscularis mucosae.

Most interesting was one experiment in which after four hours of bathing the mucous membrane with pure gastric juice Langenskiöld was puzzled at finding little sign of irritation. Investigation soon showed that in this case the animal had eaten a little meat an hour before the experiment was started. Acting on the hint derived from this accidental discovery, Langenskiöld performed another series of experiments in which he bathed the gastric and intestinal mucous membrane first with 500 c.c. of a saturated solution of Witte's peptone and then with the acid. As he had expected, he found that the presence of peptone in the tissues protected them from digestion. Similar observations have recently been made by Mann and Bollman who took dogs with a gastric fistula and injected 0.4 per cent hydrochloric acid, drop by drop for eight hours a day. After two weeks the resistance of the mucosa to this insult seemed to fail, and the animals rapidly lost weight. In another two weeks a chronic ulcer could usually be found near the lesser curvature. The presence of this lesion made the mucous membrane even less resistant to injury by acid than it was before, and the stomach and duodenum were made highly sensitive as shown by the tendency of the animal to vomit as soon as the experiment was started.

Most interesting is the fact that if the dog was given a little food before the daily experiment started, and even if the remnants from this food were later washed out,

the gastric mucosa was resistant to the harmful action of the acid, and what is more, it rapidly neutralized the acid. These experiments are of tremendous importance because they show the physician that when he gives food at frequent intervals to a patient with ulcer, this food serves not only to sop up the gastric acid but it does something to the mucous membrane which serves to protect it from injury. What we physicians have learned to do empirically is now shown experimentally to be based on a firm foundation.

As is well known, inactivated pancreatic juice is inert, and according to Matthes and others, the active form of trypsin does not ordinarily attack living cells. I have not yet been able to find much evidence to indicate that the pancreatic juice causes ulcers.

There is ample evidence to show that the shutting off of the circulation to cells promptly produces changes in them which make them subject to digestion by ferments. Ivy was particularly impressed in his experimental work with the fact that inanition or cachexia in an animal predisposes some of its cells to digestion and leads at times to the formation of ulcers. Other experimenters and some clinicians have commented on the deleterious effects of anemia in the healing of ulcers. Somewhat against this view is the fact that Ewald tried in vain to produce ulcers by feeding hydrochloric acid to anemic dogs.

It may be of interest to note that not infrequently the drinking of much cold water by an overheated mountaineer will give rise to painful symptoms which suggest the presence of an acute ulcer. It is possible that the cooling of the cells in the mucosa injures them to such an extent that later they succumb to the digestive action of the gastric juice.

FIRST CHRONIC ULCERS DISCOVERED IN ANIMALS

As was pointed out early in this article, almost all of the ulcers produced by the first experimenters were shallow lesions or hemorrhagic patches which soon healed. In a few cases typical, chronic, punched-out, bleeding, or perforating and death-dealing ulcers were discovered, usually after operations designed to divert the bile or the pancreatic juice from the duodenum (Whipple, Ivy and Fauley, Jona, Kapsinow, Gallagher, Elman and others) (Fig. 1 *o*, *p*, *q*, *r*). Bollman and Mann discovered chronic duodenal ulcers in many dogs with a true Eck fistula, and particularly in those which were given a coarse diet.

In 1903 Watts, while studying gastroenterostomy in dogs, lost one of his animals with a perforated jejunal ulcer, and since then a number of experimenters have had a similar experience. Dott and Lim, Montgomery and others found them occasionally after gastroenterostomy with exclusion of the pylorus, and surgeons soon learned that this type of operation (v. Eiselsberg's) must not be done on account of the high percentage of jejunal ulcers which appear later. (Fig. 2 *m*.)

In 1908 Borszeky produced a perforating jejunal ulcer in a dog operated on with a Y type of gastroenterostomy and given hydrochloric acid, and in 1911 Exalto had similar success with five of seven dogs operated on with the Y operation (Fig. 2 *j*). Exalto realized that this type of gastroenterostomy is dangerous because the jejunum at the anastomosis with the stomach is not bathed in the neutralizing secretions which normally come down from the duodenum.

Later Exalto, von Roojen and others gathered statistics to show that in man the number of jejunal ulcers seen after the Y operation is so great that it should seldom be used. A number of surgeons then became afraid of anterior gastroenterostomy with enteroanastomosis because with this operation the duodenal juices do not have to go past the orifice in the stomach (Levai). One would think that the question of safety or danger in this operation could be settled quickly by an appeal to statistics, but as Balfour has pointed out, surgeons today perform an-

terior gastroenterostomy only when adverse conditions make it impossible for them to do anything else, and naturally the results ought to be worse than those secured with a posterior anastomosis and a short loop. (Fig. 2 *k*.)

To avoid this objection one has to go back to old statistics gathered in the days when anterior gastroenterostomy was the only operation used. Rather impressive in this regard is the paper of Schwarz in 1914 who recorded that before 1908, with a comparatively small series of cases in which anterior gastroenterostomy and enteroanastomosis had been performed, nine jejunal ulcers occurred. He then changed to the modern type of posterior gastroenterostomy and in his next 167 cases had only one jejunal ulcer.

A number of other surgeons have claimed that the percentage of jejunal ulcers obtained with anterior gastroenterostomy is high (Wright) but others say this is not proved, and von Redwitz and Fuss, who searched the literature on this point decided that the operation has given results comparable with those obtained with posterior gastroenterostomy. Montgomery, in a number of dogs, made an anastomosis between the afferent and efferent loops and obtained only one jejunal ulcer. So far as his experience with animals went, he was not impressed with the danger of doing this operation.

A number of early investigators tried to produce ulcers in dogs by giving hydrochloric acid daily but they did not have much success. As Mann and Bollman have now shown, the acid must trickle through the stomach for hours and days until the resistance of the mucosa is exhausted. Matthes found that giving acid would delay the healing of an ulcer produced by injury to the mucosa. As will be seen later, striking results were obtained when a strong gastric juice was produced through daily stimulation of the vagus (Stahnke).

Most interesting of the earlier chance observations on ulcer is that of Bickel who in 1909 operated on two dogs (Fig. 1, *d*). Wishing to remove the duodenum, he closed the pylorus, performed gastroenterostomy, and then brought the biliary and pancreatic ducts out to the skin. A month later one of the animals died from peritonitis due to perforation of one of several ulcers which had formed in the upper part of the jejunum. Bickel promptly recognized the fact that the presence of bile and pancreatic juice in the upper part of the duodenum might be one of the most important factors in preventing the formation of jejunal ulcers after gastroenterostomy.

Realizing the value of this work, Langenskiöld, in 1914, severed the duodenum a short distance from the stomach and closed the distal end (Fig. 1, *b*). He then cut the jejunum across several centimeters below the duodenum and joined the aboral end to the stump of the duodenum. The oral end of the jejunum was anastomosed with a loop of jejunum farther caudad, thus shunting the pancreatic and biliary secretions into the middle of the small intestine. A month later, as the animal appeared to be ill, it was killed, and an ulcer was found in the short segment of duodenum joining stomach and jejunum.

Already in 1898 Chlumsky was reporting somewhat similar experiments in which he threw all the duodenal secretions into the fundus of a gastroenterostomized stomach. His animals did badly and died, perhaps before ulcers could form. He later modified the experiment so as to drain the bile into a loop of jejunum and allow only pancreatic juice to go through the stomach (Fig. 1, *c*). In 1900 he repeated Steudel's work, closing the end of the duodenum and making a gastroenterostomy so that all the duodenal juice would have to go back through the stomach to the jejunum (Fig. 1, *b*). Most of the dogs died after a few days and ulcers were not observed.

In 1910 Moutier made a total gastric pouch in a number of dogs for the purpose of obtaining gastric juice and three of the animals died with a perforated gastric ulcer (Fig. 1 *e*). He made the further

interesting and possibly significant observation that in these stomachs through which food did not pass, the ulcers did not show the usual predilection for the lesser curvature.

In 1914, Kehrer, in fifteen dogs, tied the common bile duct and anastomosed the gall-bladder and the pancreatic ducts with a loop of ileum. (Fig. 1 *i*.) Three of the animals died later with superficial hemorrhagic lesions of the gastric mucosa, and three with definite gastric ulcer. Strange to say, only once did he find an ulcer in the duodenum.

Keppich, in 1921, repeated Chlumsky's experiment of throwing all of the duodenal secretions into the fundus of a gastroenterostomized stomach. In two dogs in which 6 cm. of the pars pylorica was left in connection with the duodenum deep jejunal ulcers formed (Fig. 1 *k*). In two other animals the operation was modified to the extent that much of the distal end of the stomach was resected. In neither of these dogs did an ulcer appear. In two other animals he combined Chlumsky's and Langenskiöld's operations so as to throw the duodenal juices into the fundus of a stomach which emptied through the pylorus, into a short segment of duodenum and then into the jejunum (Fig. 1, *l*). An ulcer did not form. From his work Keppich concluded that a mixture of pyloric and duodenal secretions is more likely to produce jejunal ulcer than are the duodenal secretions alone.

Koennecke, in 1922, tested this theory by isolating the pars pylorica and first portion of the duodenum (apparently above the openings of the pancreatic and biliary ducts), closing both ends and draining this segment into a loop of ileum. The main portion of the stomach was then drained into the second portion of the duodenum as in a Billroth 1 operation (Fig. 1 *m*). Ulcers did not form anywhere. When in addition to this operation he cut the splanchnic nerves on both sides he obtained chronic duodenal ulcers in all four animals. Splanchnicotomy alone rarely produced ulcers. It is hard to interpret these results.

FIRST CHRONIC ULCERS PRODUCED AT WILL

In 1923 Mann and Williamson published the results of experiments similar to the one of Langenskiöld which, for the first time, gave the scientific world a technique which could be counted on to produce, in 95 per cent of the dogs operated on, typical punched-out chronic duodenal or jejunal ulcers similar to those found in men and women (Fig. 1, *s*). They isolated the duodenum by cutting it away from the pylorus and the jejunum. The oral end of the duodenum was closed and the distal end anastomosed with a loop of ileum so as to throw the pancreatic and biliary secretions into the lower part of the bowel. The cut end of the jejunum was then connected with the stomach at the pylorus. In most of the dogs so operated on, ulcers formed in the first few centimeters of jejunum next to the stomach. In some series of experiments ulcers developed in every animal. This experiment and modifications of it have since been performed by surgeons all over the world and with the same results.

COMPARATIVE SUSCEPTIBILITY OF PARTS OF THE DIGESTIVE TRACT TO ULCER

As already stated, a number of investigators have found that experimental ulcers made on the lesser curvature of a dog's stomach heal more slowly than those made on the greater curvature. When Morton drained the duodenum into the ileum, ulcers made along the greater curvature still healed, but those on the lesser curvature became chronic (Fig. 2, *b*). When in the animals with ulcers on the lesser curvature he performed gastroenterostomy, the gastric ulcers healed but a new one formed in the jejunum. Apparently the gastroenterostomy removed some sort of strain from the lesser curvature but it did not deprive the gastric juice of its ability to produce ulcers. In man I have several times seen gastric ulcers which

apparently developed long after the performance of a gastroenterostomy which had served to heal a duodenal ulcer.

According to Jones, and Jones and Ivy, the neutralizing action of hepatic bile and pancreatic juice is probably due to one and the same substance; that is, sodium bicarbonate. The pancreatic secretion however, is a better buffer than is the bile. According to Mann and Bollman, more important yet in protecting against ulcer formation is the neutralizing power of the duodenal mucosa. This is so strong that when the pancreatic and biliary secretions are drained away from the duodenum ulcers formed in only half the dogs operated on.

It has long been known that a Meckel's diverticulum will occasionally contain a patch of gastric mucous membrane which will produce enough juice to make a perforating or bleeding ulcer (see Aschner and Karelitz). This ulcer does not form in the patch of gastric mucosa but in the intestinal type of mucous membrane on its margins. In trying to duplicate this condition experimentally, Matthews transplanted small Pavlov pouches of gastric wall into the ileum and produced the expected ulcers.

Langenskiöld could not satisfy himself that the upper part of the small bowel is more resistant to the effects of gastric juice than the lower part, but much evidence has since accumulated to show that it is. Years ago Matthes showed that the ileum is more susceptible to injury by hydrochloric acid than is the jejunum or duodenum. Later McCann and Ivy and Fauley modified the Mann and Williamson operation, leaving a small segment of duodenum interposed between stomach and jejunum (Fig. 2, *f*). In the animals so treated the ulcers formed in the jejunum, showing that it is more susceptible to injury than is the duodenum, and indicating that this factor of susceptibility is sometimes more important than that of the "motor drive." Mann and Bollman showed a definite gradient in susceptibility to ulcer when they drained the bile and the pancreatic juice into the ileum and then transplanted loops of jejunum and ileum into the usual position of the duodenum. The duodenum was least subject to ulcer, and the ileum most subject. A similar gradation in susceptibility was found by Harper when, with the technique of Goldberg, he found that with a piece of ileum attached to a gastric pouch the ulcer which formed in the bowel perforated about the twenty-third day; with a piece of jejunum the interval was around seventy days, and with a piece of duodenum there was no perforation (Harper) (Fig. 2, *b*).

The rarity with which ulcers occur in the second and third portions of the duodenum suggests that this part of the bowel has a considerable degree of immunity to injury by gastric juice. It is possible however, that if in man the first portion of the duodenum could more easily be excised ulcers would more often be found postoperatively, distal to the line of section. Actually they have been observed in some cases after the Billroth 1 type of operation (Fig. 2, *n*).

After all this experimental work which demonstrates the great susceptibility of the bowel to unneutralized gastric juice, the puzzling, and as yet seemingly irreconcilable fact remains that when bits of gut are used as transplants in the fundus and anterior wall of the stomach, ulcers rarely develop. Perhaps the most extensive experiment along this line was done by Fiori years ago when in six dogs he transplanted a cuff of jejunum or colon into the middle of the stomach much as a plumber might insert a short section of pipe between two others (Fig. 1, *g*). Ulcers did not appear. Apparently, then, some extra factor enters in when the juice leaves the stomach to run into a loop of bowel.

It is a peculiar fact that duodenal and jejunal ulcers are usually found immediately outside the stomach where the mixture of food and acid juice first impinges. The acidity of gastric juice is rapidly lowered as soon as it comes in contact with the bowel but one would hardly expect its

digestive power to be lost before it has gone 4 cm. Actually, Langenskiöld found that the acidity of gastric juice was not entirely lost even after two or three hours of contact with the duodenal mucosa. It will be remembered also that when gastrojejunal ulcers form they are usually to be found in the distal loop which carries food away from the stoma. I wonder if possibly in some cases the duodenal juices fail to bathe the distal loop properly because food from the stomach, entering the proximal loop, tends to block the aboral passage of peristaltic waves. If it were not for an increase in the complexity of the operation the ideal technique for gastroenterostomy might be one in which the jejunum was cut across, the distal end anastomosed with the stomach and the proximal end connected with the side of the distal end 2 or 3 cm. distant from the stomach (Fig. 2, *r*). This would insure the bathing of the danger spot with duodenal juices.

INFLUENCE OF TRAUMA

This was emphasized by Baggio who folded the anterior wall of the stomach so as to make a ridge against which food would rub. He found later that erosions and sometimes ulcers formed either on the summit of the fold or on the posterior wall opposite it. This observation is in agreement with that of pathologists who often find an ulcer on the summit of a benign gastric tumor.

That trauma due to the passage of food over the mucosa is not necessary to the formation of ulcer was shown by the work of Moutier with pouches of the whole stomach (Fig. 1, *e*). It is shown also by the fact that ulcers develop in Meckel's diverticula, away from the current of food, and it has been shown recently by Goldberg and Harper. A few years ago Mann and Bollman found that a fistula into the digestive tract would not leak when they interposed between the stomach or bowel and the skin a short segment of intestine so oriented that peristalsis was directed toward the viscus. When Goldberg and Harper connected such a fistula with a pouch made from the fundus of the stomach of a dog they found that always there appeared a deep perforating ulcer in the bowel adjacent to the gastric mucosa (Fig. 2, *b*). That stagnation of juice is an important factor in the causation of the ulcer is shown by the fact that the lesion does not form if the fistula is made so that peristalsis in the loop is directed toward the skin. Incidentally, this discovery now furnishes investigators with an ideal method for estimating the value of measures designed for the healing of ulcers.

It is hard enough to explain ulcers in the efferent jejunal loop on the basis of trauma and poor neutralization of gastric juice but it is still harder to explain the occasional appearance of ulcers in the gastric side of the stoma.

A curious puzzle in regard to the mechanism underlying ulcer formation in the Mann-Williamson experiment is to be found in the fact that when the distal end of the closed off duodenum is anastomosed with the pars pylorica or fundus so that the pancreatic and biliary secretions have to go through the stomach on the way to the jejunum, jejunal ulcers form almost as often as they do when the duodenal juices are shunted into the ileum (Fig. 2, *e*). This type of experiment, first performed by Chlumsky, Schmilinsky, Enderlen et al., and Keppich has, in recent years, been repeated many times by McCann and by Ivy and Fauley (Fig. 1 *a*, *j*, *l*; Fig. 2, *e*). The failure of the duodenal secretions to protect when they are thrown into the stomach may be due to the curious fact, shown by McCann with fractional tests, that the procedure does not appreciably modify the curve of gastric acidity.

Incidentally these experiments offer no support for the belief of a number of surgeons that cholecystgastrostomy is a good operation for the cure of ulcer. In dogs it is a very bad operation because it leads to infection of the liver and subsequent marked destruction of that organ.

WHAT DERANGES THE NORMAL PROTECTIVE MECHANISM AGAINST ULCER?

The next question is: In the patient with ulcer, what has happened to upset the mechanisms which normally prevent the gastric juice from eating into the mucosa? As yet we do not know, but the research work which has been done now enables us to make some guesses. In the first place many physicians and surgeons have been impressed during the last ninety years with the importance of nervous and psychic factors. A man whose ulcer has been healed, or at least quiescent for years, will perhaps experience some psychic shock; he will be upset by some crisis in his love life or his business, and within a few hours he will be suffering again with hunger pain. I remember well an inventor who was laid low with a gastric hemorrhage every time he received a telegram stating that important machinery on which his income depended had failed under certain tests and would have to be redesigned. The German literature contains references to many such cases, as for instance, to the lawyer who had a hemorrhage every time he tried a case, or to the woman who bled from the stomach when she heard that her husband had been severely wounded at the front.

Those physicians who are inclined to look on the recognition of psychic factors in the production of ulcer as a recent development, dating back at most to the work of von Bergmann, Westphal, Westphal and Katsch, and others in 1913, will doubtless be surprised to learn from Hauser that Siebert wrote on the subject in 1842 and Grünsberg in 1852.

Similarly, those who were interested last year in Cushing's statement that ulcer may sometimes be due to disease in or injury to certain parts of the brain may be surprised to learn from von Redwitz and Fuss (p. 152) that Schiff wrote on this subject in 1846 and that in 1828 Camerer was producing ulcers in rabbits by injuring the vagus and splanchnic nerves.

It is an interesting observation, reported by many pathologists, that ulcer is seen fairly commonly in patients dying with brain tumor. Interesting also is the observation of Robertson and Hargis and others that ulcer is common in patients dying with exophthalmic goiter, a disease which is associated with great irritability of the nervous system. Strange to say most observers have found that in such patients the gastric acidity is lower than normal.

INFLUENCE OF NERVOUS TENSION

Granting that psychic influences are important and that ulcers commonly appear in persons who live under nervous tension, the next question is: How do these nervous influences act? First, there is the established fact that psychic stimuli of a certain type increase the acidity and peptic activity of the digestive juice. Second, it is possible that smaller amounts of the mucus and other protective fluids of the stomach may be secreted. Third, ischemia of the tissues may be produced by spasm of the blood vessels or of the muscularis mucosae or of the main muscular wall of stomach and bowel. Fourth, when it is remembered that the surest way in which to produce ulcers is to shut off the pancreatic and biliary secretions, one must wonder if ulcers are not being produced by an emotional drying up of these secretions similar to the drying of the saliva in the mouth. Fifth, there is the established fact that emotion can produce pylorospasm with stagnation of gastric contents and resultant increase in acidity. Might there not also be spasm and reverse peristalsis in the third portion of the duodenum or first portion of the jejunum producing harmful duodenal stagnation? These suggestions will now be taken up one by one to see what experimental or clinical observations can be brought forward to their support.

It has long been known that the gastric juice of some nervous persons is markedly acid. It has not been so well known that

the amount of pepsin is sometimes greatly increased until it is twenty or thirty times normal. Perhaps the greatest difficulty with the patients with intractable ulcer or with a tendency to the formation of ulcers is that the gastric cells go on secreting acid at times when there is no food in the stomach to sop it up. In many cases the stimulus arises in the ulcer, and the stomach keeps secreting fluid much as an eye keeps watering when the cornea is injured. This is shown by the fact that an ulcer in a Pavloff pouch will cause it to secrete excessively. Similarly, in Harper's experiment, as the ulcer forms in the segment of bowel leading from the gastric pouch, the acidity of the pouch juice progressively increases (Fig. 2, *b*).

In some patients the stimulus may keep coming to the stomach from an overly active and irritable brain. In this connection the extensive work of Stahnke is interesting. He showed in dogs that electric stimulation of the vagus nerves for forty minutes a day for periods of from two to three months resulted in, first, some increase in gastric acidity, second, a marked and often five-fold increase in peptic activity, and third, the occasional production of ulcer.

It is interesting to note also that ulcers can be produced in the stomach of a dog by the manoeuver of sham feeding (Silbermann). The esophagus is brought out in the neck so that the food drops to the floor as soon as it is eaten. Animals of a certain temperament will snap it up again and go on eating greedily for perhaps three-quarters of an hour. The result is a powerful stimulation of the gastric glands; large amounts of fluid of high acid and peptic titer are secreted, and an ulcer sometimes forms in the stomach.

Hemorrhagic erosions and possibly ulcers have been produced also by the giving of toxic doses of pilocarpin, nicotin, neurin and other drugs designed to stimulate the gastric glands by way of the nervous system. It is probable that the bad effects of tobacco on the symptoms of ulcer in man are due to some irritant influence of the drug on the nervous system.

Some persons have wondered if ulcer might not be a sort of Raynaud's disease of the stomach and duodenum. Against this theory is the fact that when Stahnke stimulated the vagus nerves he found that the blood vessels became widened and the stomach redder than normal. It seemed appropriate to him that nervous influences which greatly increase the activity of the stomach should, at the same time, help the tissues to obtain the necessary increase in blood supply.

It is probable, however that many psychic and emotional disturbances affect the bowel also by way of the splanchnic nerves and these might produce a shut-off in circulation. Actually Barcroft has noticed blanching of the mucous membrane of "exteriorized" segments of bowel transplanted into the abdominal wall, and it is still possible that vascular spasm has something to do with flare-ups in ulcer symptoms. Unfortunately as yet no one knows much about the relative importance of the sympathetic and parasympathetic divisions of the nervous system in producing gastrointestinal disturbances. It is doubtful if the drugs that have been used in an effort to distinguish vagotonia from sympathicotonia have any value; the trouble is that most persons who are overly sensitive to one group are overly sensitive also to the other. Much of what has been written on the subject is of doubtful value.

During all his work, Stahnke watched the stomachs of the dogs with the roentgen ray and at no time did he see spasm of the muscle so prolonged that it could conceivably produce ulcer. Other investigators have seen spasm in the muscularis mucosae which they thought might shut off the circulation of the mucous membrane.

There are reasons for believing that powerful psychic stimuli might dry up the pancreatic and biliary secretions. Everyone knows that emotion can stop the saliva from flowing, and the experiments of Beaumont on the stomach and of Mac-

ewen on the cecum have shown that the gastric and intestinal mucous membrane will become dry under the influence of anger or worry. In the case of Alexis St. Martin, fear, anger, or whatever depressed or disturbed the nervous system caused the villous coat of the stomach to lose its smooth and healthy appearance; it became sometimes red and dry, and at other times pale and moist; the secretions became vitiated, greatly diminished, or entirely suppressed, and the layer of mucus became scarcely perceptible.

Suggestive of the presence of pancreatic dysfunction of psychic origin are the pale, bulky, fatty stools sometimes passed by sensitive persons who have eaten under the influence of excitement, fear or anger.

DUODENAL STASIS

He who remembers how commonly nausea and vomiting result from mental impressions will be inclined to wonder if psychic influences might not reverse somewhat the peristaltic trends in the upper part of the digestive tract, at least enough to produce temporary stagnation of gastric contents in the duodenum. His idea would be that such stagnation might possibly upset the mechanisms that protect this part of the bowel from the formation of ulcers. Sloan has reported many experiences which convinced him that not infrequently duodenal ulcer or pseudo-ulcer can be cured simply by loosening adhesions about the ligament of Treitz and thereby giving better drainage to the duodenum. According to Hurst and Stewart (p. 67) duodenal stasis is not infrequently associated with duodenal or gastric ulcer, and Slocumb has collected much evidence to strengthen this contention. Actually, I think the impression of most roentgenologists is that in cases of duodenal ulcer the duodenum tends to empty quickly. Somewhat against the theory that duodenal stasis might favor the formation of ulcers is the fact that they rarely develop during pregnancy when there is much back-pressure in the digestive tube; in fact there are records of cases in which pregnancy seemed to serve as a cure for ulcer.

PRESSURE EXERTED BY THE ABDOMINAL WALL ON THE ULCER

While talking about duodenal stasis as a possible factor in the production of ulcer it may be well to mention the curious fact that many patients with ulcer or pseudo-ulcer are distressed if they bend over so as to put pressure on the organs in the upper part of the abdomen, and they are relieved when they sit upright or bend a little backward. Long ago my attention was called to this when an intelligent man who had suffered for twenty years with ulcer told me that he has been cured by lowering his chair at the office so he wouldn't have to bend over his desk all day. Later I found that some of my physician friends with pseudo-ulcer promptly get pain if when driving an automobile they sit hunched over the wheel. Others get pain if they bend over to weed a lawn. The literature contains many references to this phenomenon, and German writers speak of the frequency with which an ulcer type of pain is met with in tailors, shoemakers, and others who sit bent over at their work.

According to a number of writers, this distress is due to pressure brought to bear by the principal fold of the anterior abdominal wall on the stomach or duodenum. The phenomenon was first described by Rasmussen in 1887 and it was later studied in detail by Hitzenberger and Reich, Rohde, Schwarz and others. It has even been suggested that in women, with their tendency to ptosis, the fold presses on the middle portion of the stomach and causes gastric ulcer, while in men with a high stomach it presses on the duodenum and causes duodenal ulcer.

It is well known to every clinician that lifting or straining is harmful to patients with ulcer. I often tell them that the stomach is like a hammock that hangs from two hooks and one of these hooks contains the ulcer. Obviously, any move-

ment that puts strain on the hook is likely to make the ulcer worse, and I have seen a number of patients in whom lifting promptly brought on a hemorrhage.

PROBLEMS OF TREATMENT

In young patients with ulcer, and in the many patients with pseudo-ulcer the great desideratum is some method of overcoming the predisposition to the disease. Many patients doubtless would be well if they could live an easier life, both mentally and physically, and many could probably get more satisfactory relief through the giving up of ambitions and living simply than they get from expensive courses of treatment in one hospital after another. A few clinicians already know that in some cases they can get as good results with nerve sedatives, soporifics and rest as with alkalies and diet; and the taking of an hour's rest after each meal will sometimes help wonderfully. In cases of pseudo-ulcer a vacation usually works like a charm, and often with real ulcer it will bring about a remission of symptoms.

Active research must be continued to find some method of lowering the digestive power of the gastric juice. One approach to the problem would be through a study of gastric secretion in young persons with achlorhydria. If we could learn something about the mechanism that produces this abnormality we might be able to imitate it in patients with ulcer. So far, fat has been the secretory depressant most widely used by the clinician. It has the double advantage that it lowers acidity and it tends to remain long in the stomach. If it left rapidly its effects would be too transient. I think Glassner's and Hurst's and Stewart's (p. 393) method of giving a little olive oil or some other fat at bedtime deserves further trial and careful study. Much fat cannot be used during the day because it causes nausea.

It has long been known, of course, that the corner stone of any medical treatment for ulcer must be the giving of food at frequent intervals. The explanation usually given to patients is that the food sops up the acid and combines with it. Perhaps even more important is the protective action exerted by absorbed peptones and other food substances on the cells in the mucosa. This discovery of Langenskiöld and Mann and Bollman is of tremendous importance to the clinician who now has even more reason than he had before to urge patients with ulcer to keep food in the stomach.

Furthermore there is every reason now to advise the patient after gastroenterostomy to continue for several weeks with frequent feedings until his gastric and intestinal mucous membranes have lost their sensitiveness to acid and until much of the danger from jejunal ulcer has passed. Actually if we take Mann and Bollman's observations together with those of Palmer on ulcer pain, we can be fairly sure that with some duodenal ulcers and at certain times in every patient with ulcer there is a marked sensitization of the gastric mucosa. It may well be that the presence or absence of this sensitization has much to do with determining whether gastroenterostomy will be followed promptly by the development of a jejunal ulcer.

If I had to submit to the resection of a florid jejunal ulcer I think I would want the surgeon to sew in a catheter with its tip near the stoma and to start immediately with a drip consisting of a solution of peptone. I would want this kept up for a few weeks until the mucous membranes had lost their sensitiveness to acid, and with it, perhaps, most of their susceptibility to recurrent ulceration.

The effects of atropin are usually transient; I have no confidence in them, and often the treatment is more annoying than the disease. Mann has not been able to heal experimental ulcers with it. As Hardt and Rivers showed years ago, the development of alkalosis imposes a limit to the use of alkalies, and as Crohn and, more recently, Harper have shown, there is a strong tendency to a rebound when the acid is forced down by neutralization. In some patients and in many dogs gastric

acidity just cannot be controlled even during the day. Injections of foreign protein, while clinically helpful in some cases, do not consistently lower gastric acidity (Vanzant).

Probably one of the greatest faults of our present method of treating ulcer is to be found in the fact that nothing is done to neutralize the acid secreted during the night. Winkelstein has tried to obviate this by feeding the patient continuously through a small indwelling tube but the discomfort and interference with sleep is often so great that I doubt if the method can ever come into general use.

During the last two years many physicians, following the lead of Fogelson, have been experimenting with mucin, and it is possible that something will come of this work. Unfortunately mucin is not a particularly good buffer for hydrochloric acid, and what little effect it has is apparently due to the peptone which is mixed with it (Jones and Ivy, Mitchell, Vanzant). Moreover, the mucin made thus far has been unpleasant to take and many patients have refused to continue its use. With a better product some of these objections may be overcome.

TYPES OF OPERATION USED

Gastroenterostomy is still the best and most practical operation available for the cure of duodenal ulcer; in eight or nine cases out of ten it works like a charm and the patient is well. Unfortunately in two or three cases out of a hundred the patient returns with a jejunal ulcer, and in an occasional one of these patients a new ulcer will form almost as fast as the old one is excised.

In their desire to protect patients from this danger some surgeons have turned to the operation of subtotal gastrectomy. In Germany, with the complicated type of ulcer that is seen so commonly, this operation is doubtless often the only one that can be counted on to work a cure, but in most of the cases seen in the United States it does not seem right to do such a big operation or one with a risk so much higher than that of gastroenterostomy. It would not be so bad if subtotal gastrectomy would always safeguard the patient from jejunal ulcer, but unfortunately it does not do this. It appears to do it in cases in which an achlorhydria is produced, but unfortunately this object is not always attained even when more than half the stomach is removed. Similarly the Billroth II operation does not safeguard the patient from the formation of a jejunal ulcer (Fig. 2, *o*).

The Billroth I type of operation, or an excision of the ulcer with pyloroplasty does not carry with it the dangers incident to the formation of a jejunal ulcer, but unfortunately these operations can be performed only in certain favorable cases (Fig. 2, *n*). In some cases, also, the relief obtained by the patient is not so complete as it commonly is after gastroenterostomy.

AVOIDANCE OF JEJUNAL ULCER

How can we physicians and surgeons hope to pick out some day the patients in whom an operation and particularly gastroenterostomy had better not be done? I can conceive of at least two ways in which this end might be accomplished: one is through an anthropologic type of study of the individual and his reactions, and the other is through a study of his gastric juice and the way in which it is secreted. For several years we at The Mayo Clinic have been becoming more and more fearful of operating on the keen, nervous, active, hard-living, "go-getter," sales-manager type of man, and especially when he is of Jewish ancestry. The Jew seems to be particularly subject to a severe type of recurrent ulcer, so much so that at Mt. Sinai Hospital in New York Berg and his colleagues have given up the operation of gastroenterostomy in cases of duodenal ulcer and have substituted for it extensive resection of the stomach combined with the cutting of one vagus nerve.

Years ago von Bergmann, Westphal, Westphal and Katsch, and others in Germany worked out a large series of tests which they applied to persons with ulcer to show that most of them have hypersensitive nerves and signs of marked vagotonia, but unfortunately they were not impressed with the need for first applying the tests to a control group of persons without ulcer. The idea back of their work seems to be a good one, and for years I have been trying to get someone trained in the field bordering on physiology, psychology and anthropology to study a large group of persons: some normal, some with nervousness of various kinds, some with pseudo-ulcer, some with easily controlled ulcer, and some with uncontrollable or "dysiatric" ulcer, to see if there is anything peculiar about the nervous and psychic reactions of the persons in the several groups. A particular effort should, of course, be made to see if there is anything distinctive about the physical or mental or nervous makeup of the patient who is highly subject to the formation of jejunal ulcers (Winkelstein).

We may also find some way of recognizing persons highly subject to ulcer through the study of peculiarities in the gastric juice and the ways in which it is secreted. We know, of course, that patients with a high acidity which is not easily controlled by diet, alkalies, or rest in bed are likely to give trouble after any form of operation. But often this criterion fails, and patients with a strong tendency to the formation of ulcers will have only a moderate increase in acidity. Recently Vanzant et al. found that most members of a group of patients with a severe and intractable type of duodenal ulcer had concentrations of pepsin several times the normal. From the point of view of the diagnostician who is often baffled because the distributions of measurements of acidity from normal and diseased persons overlap, the encouraging feature about this work is that while a patient with severe ulcer may have a free acid titer of 60, well within the range of normal variation, his pepsin titer may be 1500 units instead of the 90 which is about normal for an average phlegmatic person.

It might be helpful to study also the reactions of the ulcer-bearing patient to sham feeding, and more might be learned about the mechanisms which cause juice to flow when the stomach is empty.

MECHANICAL FACTORS AT THE PYLORUS OR AROUND THE STOMA THAT MAY LEAD TO THE FORMATION OF JEJUNAL ULCERS

The work of Mann and his students has now made it clear that one of the most important factors, if not the most important in the production of ulcers, at least in loops of intestine draining the stomach, must be a failure of neutralization of gastric juice by the pancreatic and biliary secretions. It has also been shown time and again, both in animals and man, that any form of gastroenterostomy which interferes with the easy access of the duodenal secretions to the mucous membrane about the stoma leads to the formation of ulcers there. The most dangerous of all is gastroenterostomy with von Eiselsberg's closure of the pylorus. In some clinics one out of four patients so operated on has returned with a jejunal ulcer.

The next question is: why should closure of the pylorus lead to such dire results? In the first place, there is the strong suspicion based on the work of a number of investigators, that the gastric chyme is not able to produce so good a stimulus to pancreatic secretion when it comes in contact with the jejunal mucosa as when it is poured normally into the duodenum. Burdenko found in a few dogs with gastroenterostomy, pyloric closure, and a pancreatic fistula that the giving of food produced very little juice: sometimes only a fifth or an eighth of the normal amount, and at necropsy there was an atrophy of the pancreas. Enderlen et al. also made a fistula into the duodenum and found very little secretion there after pyloric exclusion.

The next possibility is that with the closure of the pylorus the small amount of

pancreatic and biliary secretion which is poured out is not pushed on into the jejunum. Years ago Wheelon and Thomas, and Mahoney and I showed that the peristaltic waves that sweep material through the duodenum usually start about the time that a little chyme is pushed through the pylorus by a gastric wave. There is reason to believe, then, that duodenal peristalsis should be weakened by closure of the pylorus and particularly by a complete separation of stomach from duodenum; actually there is experimental evidence to show that this does happen (Enderlen et al.). Enderlen and his coworkers found that the juices stagnating in the duodenum after pyloric closure not only possess great digestive power but their presence serves to prolong the third, or intestinal phase of gastric secretion. Obviously, this is highly undesirable.

Theoretically, duodenal peristalsis ought to be more nearly normal when the surgeon cuts the stomach across about 12 cm. above the pylorus so as to leave the powerfully muscled gastric antrum in connection with the bowel, and actually Balfour tells me that this operation, used by Kelling, Denk, Devine, Balfour and others, is seldom followed by jejunal ulceration (Fig. 2, *p*). That it does not absolutely protect the patient against this mishap is shown by Denk's report of two cases seen in his clinic.

Theoretically, one of the surest ways of producing a jejunal ulcer should be to close the pylorus and make a duodenal fistula, and actually, this undesired result was obtained when these operations were done on a patient whose case has been reported by Schostak. Following appendicitis the woman had a cecal fistula through which the contents of the duodenum later discharged. In order to close this fistula, gastroenterostomy was done and then a pyloric exclusion. Within five weeks after the gastroenterostomy was performed the patient began to suffer with the pain of a jejunal ulcer which soon perforated.

The next question to arise is: if a man-made pyloric closure so commonly leads to abnormal pancreatic and duodenal function with resultant production of jejunal ulcer why shouldn't the pyloric closure which is due to the presence of a large sclerosing ulcer produce equally bad results? Why shouldn't the surgeon be particularly fearful of performing gastroenterostomy in the presence of a closed pylorus? The need for a clear-cut answer to this question is so obvious that I cannot understand why some surgeon has not long since given it to us. Perhaps someone has, but in months of reading I have not yet run onto any paper in which sufficient data have been analyzed from this point of view.

The lack of curiosity on this point seems to be due to the surgeon's conviction that he gets his best and happiest results from gastroenterostomy in men who have suffered for years with an obstructing type of ulcer. There being no question about this fact, let us see if it can be accounted for in any way. Eusterman's impression is that the best results are obtained in older men in whom the obstructing ulcer has more or less burned itself out and in whom the gastric acidity is not as high as it used to be. He suggests also that, with a gradual closure of the pylorus, some adjustment may take place which, later, when gastroenterostomy is done, serves to protect against the formation of jejunal ulcer.

According to von Redwitz and Fuss it is the young patient with a florid ulcer and pyloric obstruction who gets a jejunal ulcer after gastroenterostomy. Denk also has commented on the danger of performing gastroenterostomy in the presence of an active ulcer and pyloric obstruction. In Schwarz' abstract of 130 cases of gastrojejunal ulcer I find mention of pyloric stenosis in eighty-three; in one case pyloric exclusion was done, in thirteen the lesion was a duodenal ulcer, in eleven it was a gastric ulcer, and in twenty-two no details were given. These data are suggestive, but unfortunately Schwarz did not supply

information about a control group of patients with pyloric stenosis who after gastroenterostomy did not succumb to jejunal ulcer. Actually, we must expect to find most patients with pyloric stenosis escaping gastrojejunal ulcer because we know that three out of four with complete, man-made, pyloric exclusion escape.

The question is: Is the danger large enough so that surgeons should take pains to avoid it? One would expect the pyloric closure due to scarring to be less complete than that produced intentionally by the surgeon, and possibly a small amount of chyme trickling into the duodenum is enough to maintain pancreatic function and duodenal peristalsis. Possibly the surgeon should imitate nature, and when he closes the pylorus he should leave a small hole. Perhaps in every case of obstructing ulcer in a fairly young tense person with high acids he should try to make, in addition to a gastroenterostomy, some sort of an opening between stomach and duodenum.

Obviously, of course, pyloric closure cannot be the only cause of jejunal ulceration because this complication occurs occasionally when gastroenterostomy is done for gastric ulcer or for no ulcer at all.

Eusterman's impression from a large experience is that in most cases the pylorus opens after the ulcer heals, and he doubts if gastrojejunal ulcers are more frequent in the cases in which stenosis persists. I believe, however, that it would pay someone to investigate this point statistically and with great care.

The fact that in some cases gastrojejunal ulcers tend to recur every time they are excised indicates strongly that the cause is not some defect in technique or some peculiarity in the behavior of a particular stoma or of the loops fastened to it because one would not expect these same accidents to occur with every operation. What seems more probable is that either some patients are highly susceptible to the formation of ulcers or else the fact that a jejunal ulcer has been present predisposes to the formation of another. What we need greatly is some answer to the question: why, in some patients does the concentration of acid in the stomach remain high after gastroenterostomy and even after subtotal gastrectomy?

TREATMENT

One hint in regard to treatment has come from several of the men who during recent years have been studying experimental ulcer. As I pointed out early in this paper, many laboratory workers discovered years ago that when in dogs they snipped out bits of gastric mucosa the defects healed rapidly. When Ferguson tried this in rabbits he found that some of the lesions were converted into chronic ulcers and he assumed that this was due to the constant presence of rough food in the stomach (Fig. 2, *d*). Other observers found a difference in the rate of healing of experimental ulcers depending on the smoothness or roughness of the diet, and on several occasions experimenters have lost dogs because a piece of celery or turnip poked a hole through the floor of an ulcer (Mann, McCann, Bollman). In some cases hair swallowed by the animal has served to retard healing (Bollman, Pappenheimer and Larimore).

As Mann and others have shown, when an ulcer begins to heal, the defect is covered by a layer of epithelium one cell deep. It does not take much rubbing to remove such a membrane, and it becomes obvious that whatever other qualities an ulcer diet should possess, it at least should be smooth and free from rough indigestible particles.

As I said before, experimentation suggests that more work should be done with fats and oils in the treatment of ulcer. They have been used particularly by Glaessner and Hurst but unfortunately they tend to produce nausea and hence there are limits to their use during the day. They might, however, be used at night when they are most needed to inhibit secretion.

SUMMARY

A review has been made of many observations made in the clinic, the operating room, and the laboratory, all of them throwing light on the mode of causation of peptic ulcers and on the ways in which such ulcers should, perhaps, be treated.

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THE NON-SURGICAL TREATMENT OF PEPTIC ULCER

WITH A CLINICAL CONSIDERATION OF THE RELATIVE THERAPEUTIC VALUE OF GASTRIC MUCIN*

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ACCURATE data respecting the results of non-surgical management of peptic ulcer are difficult to secure. One of the chief reasons for this dilemma is that we are not possessed of means whereby, for the individual patient, it is possible to estimate how great is that patient's natural tendency to heal. Thus it happens that so-called "absolute cure" of an ulcer may have resulted spontaneously and would have taken place irrespective of what non-surgical regime had been carried out. That spontaneous healing is not uncommon is proved by the frequent demonstration of healed ulcers at autopsy in patients who never had been aware that ulcer existed or, if they had been chronic dyspeptics, they had accepted the condition and had treated distressing symptoms by simple home remedies or with the aid of advertised medicines. Before the advent of exploratory abdominal surgery and the roentgen ray, and the popularization of peptic ulcer through lay and medical press, patients, though familiar with the discomforts accompanying gastric or duodenal ulcer, were not acquainted with its possibilities as a disease entity. They, as not uncommonly now, came to the physician for the complications of ulcer, particularly obstructions, massive hemorrhage, perforative attacks or malignancy. Sufferers they were, but when it is recalled that barely 21 per cent of *all* peptic ulcers are accompanied by stenoses or the dramatic accidents of gross hemorrhage, perforation or malignancy, one readily may understand how, with such low percentage of mortality even in the presence of chronic morbidity (fortunately, usually, intermittent), years of discomfort are endured with relatively little incapacitation, physical or economic. Ulcer subjects under my care have experienced ulcer-types of dyspepsia for an average of eleven and one-fourth years and yet fewer than 17 per cent are incapacitated or present signs of malnutrition. Often enough, the ulcer itself causes little discomfort, but bowel irritability, a complaining appendix or a sluggishly functioning gall bladder cloud the ulcer picture. When discomfort thus is localized, pain or fear lead to the examination at which ulcer is discovered. The publication of the observations of surgical pathologists concerning the relationship of gastric ulcer and gastric cancer exerted a more powerful influence towards bringing chronic ulcer-dyspeptics under medical care than did the actual discomforts and serious complications of the ulcers themselves. While this result has been a means of aiding sufferers, yet the significance of this desired state upon the healing of ulcer lesions still requires proof. Many physicians and pathologists are well aware that physical comfort does not mean that the lesion of which peptic ulcer is a manifestation has healed: all too often, one observes serious hemorrhages or fatal perforations in patients who were, until the catastrophe, absolutely symptom-free.

Medical statistics of ulcer cure are handicapped by the improper selection of material for treatment. We are acquainted with few statistics which detail the results following non-surgical management of duodenal ulcer, as distinct from those of gastric ulcer. Neither are we familiar with records which list, separately, the results of non-surgical procedures with respect to

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calloused, recurrent ulcer, ulcer producing crater deformity, free-bleeding lesions, ulcer with perforation, or (with the exception of ulcers in syphilis), ulcer accompanied by true organic stenosis.

Twenty-eight years ago, Ewald[1] refused to make statements respecting how often his non-surgical management had cured peptic ulcer. However, he reported in 1919 that Schultz had followed up 299 instances of the affection (all of which had been accompanied by gross hemorrhages) and from 157 replies had estimated that permanently good results had followed in 64 per cent. In 23 per cent the treatment was a failure and there was a mortality of 7.4 per cent.

In 1912, Friedenwald,[2] reporting upon 107 indiscriminately selected patients treated by the v. Leube and Lenhartz methods, stated that, for a five-year period or longer following treatment, an average of 75 per cent remained cured.

I have endeavored to secure information relative to the percentage of permanent cures following the v. Leube-Sippy regimen. There has been published no report stating definitely the status of treated ulcers at periods of, say three, five, seven or more years following treatment. One must be content with Sippy's unqualified but indefinite statement[3] that in "approximately 85 per cent of all cases of pyloric obstruction due to peptic ulcer, 'obstruction' disappears during the first two or three weeks under the influence of the medical treatment advocated. In the remaining 15 per cent of cases 'obstruction' is due to tissue narrowing. If the medical management described is accurately applied the 'obstruction' can be made to disappear in about one-half of these cases also." Sippy further stated that "70 per cent of the instances which were managed were the 'obstructive' type of ulcer."

More recently, R. Brown[4] has analyzed the end-results in peptic ulcer from the services at the Presbyterian Hospital, Chicago (1912–1927) inclusive). His report concerns 1224 traceable patients (75 per cent males and 25 per cent females). The ulcers were duodenal in 923 patients and gastric in 77 patients and both in 12 patients. Brown uses as a criterion of "cure," "a present status of freedom from symptoms," admitting, however, that relapse is possible in certain patients. In his series, 49.5 per cent report themselves as cured, 16.7 per cent satisfactorily improved, or a total of 66 per cent experienced good results following non-surgical management. An additional 10 per cent experienced moderate improvement, while, in 20 per cent, medical treatment failed. Brown's statistics are in close accord with those reported by Ewald, twenty-six years previously.

In our own experience,[5] employing the "physiologic rest" regime developed in my clinic (excluding from the statistics patients who had been discharged from treatment *two years or less*) of 470 patients, cessation of the ulcer process occurred in 361 or 77 per cent. Of 66 patients who came to laparotomy for numerous intra-abdominal lesions, following institution of our ulcer management, completely healed scars were demonstrated in 54, approximately 82 per cent. In the remaining 12 patients, malignancy, perforation, multiple ulcerative or active benign ulcer lesions were present. Such results definitely prove that peptic ulcer can heal if given opportunity equal to that which the surgeon gives stomachs upon whom he has performed resection, excision or enteroanastomosis. Roentgen proof of healing was shown in 94 cases by disappearance or lessening of ulcer deformity, by improved gastric emptying rate, by disappearance of local ulcer tender areas on palpation during fluoroscopy, and by absence of the inferential, motor phenomena characteristic of active ulceration.

For no proved reason, it has been assumed that non-surgical treatment favorably affecting gastric ulcer similarly influences the duodenal lesion. Controlling gastric chemism from the standpoint of neutralizing the free HCl of gastric juice

has been thought an adequate measure for duodenal ulcer healing. My "physiologic rest" regime of management goes further than controlling the free HCl of gastric juice, in that it attempts to place the ulcer-bearing area of the duodenum in a state of peristaltic rest, and thus favors tissue repair.

Very recently, Jordan and Kiefer[6] have reported upon the end-results when duodenal ulcers have been managed by the v. Leube-Sippy routine. These workers conclude that 46 per cent of duodenal ulcers so treated will have one or more recurrences within five years after "cure" (controlled from the standpoints of clinical comfort, laboratory tests and roentgen studies) has been brought about. The greatest frequency of recrudescence is within two and three years after treatment. Failures are manifested by massive hemorrhage, obstructions, epigastric distress and tenderness, reappearance of high gastric acidity and radiologic anomalies. Hinton[7] draws similar conclusions from a review of 324 duodenal and gastric ulcers and states that "any attempt to call a patient cured after either medical or surgical treatments, unless followed for a ten year period and frequently examined, is most misleading."

MUCIN THERAPY

In an attempt to increase the efficacy of non-surgical management of peptic ulcer (gastric and duodenal) during the past two years, and, under the inspiration and guidance of Ivy and his associates, Fogelson[8] has treated patients with a powdered gastric mucin prepared from hogs' stomachs. In published reports, Fogelson and others have dealt alike with gastric and duodenal ulcers and have not considered it necessary to treat these lesions as separate entities. Fogelson's non-surgical peptic ulcer management concerns itself largely with rendering the "corrosive action" of gastric juice innocuous by emplying gastric mucin as an "antacid," a "buffer" or as a local protectant to injured visceral lining. Fogelson (1930)[9] claims that 1 gm. of mucin will combine (and neutralize?) with 12 to 15 c.c. of N/10th HCl. He claims further, that his gastric mucin has a very low secretagogue content and does not stimulate gastric mucosa more than do other bland substances. He states that the preparation has no deleterious systemic effects, has no significance upon gastrointestinal activity, causes neither constipation nor diarrhea, is digested in the jejunum and, in the presence of free HCl, is precipitated and thus becomes effective as a mechanical protectant of injured visceral mucosae.

In drawing these conclusions, Fogelson avails himself largely of the investigations of Ivy[10] and Kim[11] and of Whitlow.[12] The former's researches demonstrate the resistance of mucin (as found in stomach pouches) to penetration by free HCl and to the apparent value of fed mucin in preventing certain animals with biliary-tract fistulae from postoperatively developing "spontaneous" duodenal ulcers. Whitlow's work tended to prove the theory that the reason why the stomachs of living animals are not "auto-digested" is because of the property of mucin, freshly metabolized and excreted, to resist the penetration (and thus, digestion) of free HCl-pepsin. While Whitlow's studies deal largely with test-tube set-ups designed to mimic gastric digestion, yet he showed that, in the living animal, when the mucous membrane is robbed of its "protective" layer of mucin, it is readily attacked by free HCl and pepsin and "digested." However, Whitlow fails to present histologic material in support of his contention that swabbing away mucus from mucosae leaves normally intact and functioning epithelium.

Employing gastric mucin, exhibited *per os*, with or without food, during 1930–31, Fogelson[13] states that he treated 113 instances of peptic ulcer, of which number 56 were in the group of "intractable" ulcer. Sixteen of these patients had experienced massive hemorrhages. In the 56 patients with ulcer of this type, Fogelson reports that 49 patients (87 per cent) were

given "complete relief from all symptoms" and were returned to "economic fitness." He further states that of the entire group of 113 patients (including the 56 "intractable" instances) there were "only 7 failures" and these failures were in the group classed as "intractable." In other words, of all the ulcer subjects treated, approximately 94 per cent were considered cured. Fogelson states further that since February, 1931, approximately 200 ulcer patients have been treated with his gastric mucin in "clinics outside of Chicago," "with 90 per cent relief of all symptoms." The relative occurrence of duodenal and gastric ulcer is not given. Although in selected instances favorable alterations in roentgenographic deformity following treatment are reported, Fogelson's main criterion of "cure" rests upon cessation of symptoms and of constitutional improvement.

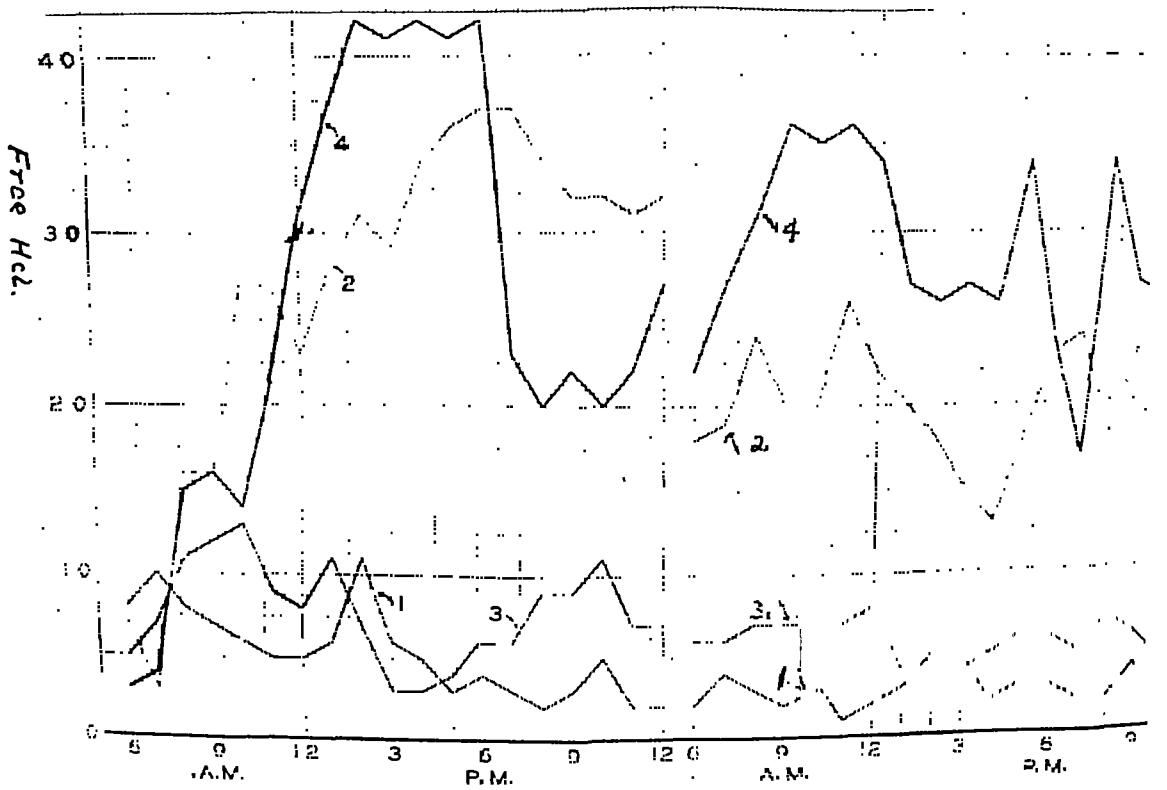
Recently, Atkinson[14] reported the results of mucin management in 43 individuals. His table only vaguely indicates the locations and types of the ulcers. In 18 patients the progress was followed by roentgen observations. Atkinson noted early relief of pain, of abdominal distress, of eructations and regurgitation and, roentgenologically, less visceral spasm and palpatory tenderness together with changes in contour. His studies of the gastric juice demonstrate that when patients were receiving mucin, the relief of symptoms bore but slight relationship to the lowering of free HCl and that the "neutralizing" or "buffering" action of mucin bore little quantitative relationship between the amount of mucin exhibited and the free HCl values of the gastric extracts. Although favorably impressed with the therapeutic value of mucin, Atkinson is non-committal with regards the worth of the preparation towards permanent cure of the ailment. In addition to receiving mucin, Atkinson's patients were on rigid diet, had rest and general care and seemingly, at times, alkali medication.

C. F. G. Brown[15] reports upon 93 patients affected with gastric or duodenal ulcers whom he has submitted to mucin therapy. He states that all his patients definitely improved subjectively and objectively on hourly feedings, general care *and* mucin. He believes that mucin management is responsible for the disappearance of pain, lessened gastric emptying-time, early cessation of bleeding and for favorable roentgen signs. Mucin only partially and irregularly affected free HCl, a transient increase commonly being followed by a lowered reading. The persistence of free HCl bore no definite relationship to the symptomatic improvement. Brown reports no control series but observes that 36 patients who had been unsuccessfully subjected to various types of non-surgical management (chiefly the Sippy regime), not including mucin, were subjectively and objectively relieved when later mucin therapy was introduced.

BIOCHEMISTRY OF MUCIN

Gastric mucin belongs to the group of "compound proteids," in the subgroup "glycoproteids." On cleavage, it yields a protein and a carbohydrate, or an acid allied thereto, but no xanthin bodies. The substance is a colloid and forms a ropy or mucilaginous mixture on attempted solution. Gastric mucin has a composition similar to that of the mucins of saliva or of connective tissue but certain poorly understood molecular arrangements make this mucin different in combining-power. When split with free HCl, acetic acid appears: strong acids break it into leucine and tyrosin. Dilute alkalies produce from it an alkali-albuminate similar to albumose or peptone in structure and reactions (Hammarsten[16]). Strong acids (25 per cent HCl) precipitate mucin and this precipitate is maintained by excess water. Pepsin has little effect upon mucin, except, perhaps, after it has been precipitated by strong acid. Trypsin readily digests gastric mucin leaving available for resorption, after the action of succus entericus, end-digestion products (amines, as tyrosin, leucine) and a carbohydrate

body (or its derived acid) which probably is stored in body tissues as glycogen. It is said (Hammarsten) that an accumulation



Curve 1. Showing, by fractional analyses, the variations in secretion of free HCl in stomach of patient with gastric ulcer under various digestive stimuli. 1, HCl secretion with subject on smooth, non-retention diet; 2, on milk, egg and cream feedings; 3, on milk, egg and cream feedings with alkali exhibited; 4, on milk, egg and cream feedings plus 180 gm. gastric mucin over a twenty-four hour period. (Author's observation.)

of these end-digestion products *hinders tryptic activity*. It is apparent that mucin is a substance of definite potentialities as a food, whether or not it serves as an "antacid," a "buffer" or a mechanical protectant to injured visceral mucosae.

PHYSIOLOGIC EFFECTS OF MUCIN INGESTION

When a "mucin meal" (75 — 100 gm. in watery suspension) is fed to the normal, empty stomach, gastric analysis returns a free HCl curve comparable to that returned when a roast beef sandwich has been eaten (Curve 1). Even should the gastric mucin-meal pass rapidly from the stomach and be cleaved in the upper intestine, the mechanical stimulation to the stomach plus the resorption of polypeptids and amino acids excites definite gastric secretion of histamine-like character, e.g. a *gastric juice in which free HCl is abundant while pepsin and nitrogenous bodies are relatively low* (Babkin[17] and Webster[18]). This type of gastric-juice response has been emphasized by Rivers, Vanzant and Essex[19] but ascribed by them to histamine-like impurities in Fogelson's gastric mucin as commercially marketed. I suggest that it is possible that such exist as split-products of mucin when gastric mucin is given therapeutically, and that here may lie an explanation of why certain favorable effects possibly may follow: *its presence (or its end-products) in excess may produce a continuous flow of an acid gastric juice, but a gastric juice of extremely low, or, when prolonged mucin exhibition occurs,*

absent, peptic power (the qualitative "histamine-produced" gastric juice of Babkin). In such circumstances, constant "corrosive," acid-pepsin attack upon damaged visceral mucosa is not possible or is lessened. The excess of split-protein products in the *duodenum and upper jejunum* likewise may interfere with tryptic attack upon injured duodenal mucosa (Hammarsten).

The researches of the Montreal physiologists upon the qualitative variations in gastric juice accordingly as that secretion is brought on by intragastric (hormonal) stimulation or by the "trophic" or nervous (vagal) route, would appear to lend support to my explanation of the manner in which favorable effects may accompany mucin therapy. Pleasant odor, attractive appearance, delectable taste or the physical character of food as served and eaten are the avenues through which "trophic" stimulation of gastric juice travels. Gastric juice *so secreted* has a peptic value 15 to 40 times of that resulting from *intragastric* or hormonal (histamine-like) stimulation. As commercially produced and when clinically administered *per os* (alone or with food), no impartial observer may claim that gastric mucin excites the pepsin-rich "trophic" flow of gastric juice. Many peptic ulcer patients are of highly organized nervous make-up. Their gastric secretions often are continuous (gastrosucchorrhea) during an entire day, and are rich in acid and the peptic enzymes ("trophic" secretion). It is of interest to observe, in passing, that in none of our patients of this "nervous" type was the exhibition of gastric mucin, therapeutically, accompanied by satisfactory clinical results. Further, in reference to the *qualitative* aspects of gastric secretion, it is common knowledge that when patients are affected with constant nausea (non-obstructive in origin), gastric juice is low both in acid and in peptic activity. In such patients, apparently *vagal* stimulation is weak. It is interesting to observe that when ulcer patients have a constant repugnance to taking gastric mucin, not infrequently clinical benefit follows, a benefit not wholly ascribable to the limited food-load to which the stomach is subjected,or to the buffer or protective qualities of mucin.

Since the secretion curves which Fogelson, Atkinson and C. Brown have exhibited, and our own observations, reveal that the "*reduction*" of free HCl in gastric juice bears little direct relation to the *quantity* of gastric mucin administered, we suggest that apart from any "buffering" or "neutralizing" effect upon the free HCl, gastric mucin may exert the favorable effect claimed by its limitation of pepsin production and by its antitryptic properties (through its end-digestion products).

PHYSICAL PROPERTIES OF MUCIN

Fogelson's "gastric mucin" is a sticky, grayish-white powder, with a somewhat offensive odor which becomes more pronounced when the preparation is moist or not kept cold. In such circumstances, the odor not rarely is fecal. Patients state that the taste of mucin corresponds closely with its odor. Ambulatory patients find it impracticable to preserve the powdered mucin under ideal conditions and hence have a growing repugnance to taking it as a medicine. C. Elliott[21] states that in his experience "it has been difficult to get patients to take it (gastric mucin) unless they were in dire need of clinical relief."

Fogelson recommends that gastric mucin be given intimately mixed with such foods as egg-nogg, chocolate malted milk, cocoa, cream soups, or thin custards. However, it has not been our experience that even when the greatest care was taken, by experts, intimately to "fold in" the mucin with food, the resultant mixture was unattractive in appearance, taste or odor. Often the mixtures are slimy or lumpy, so much so that patients may refuse the feedings and their general nourishment become impaired by partial starvation. Particularly were the preparations poorly borne by patients who experienced regurgitation of gastric contents or who frequently

belched. The odor of these regurgitated or vomited stomach-contents not rarely resembled that present in instances of fecal vomiting caused by high intestinal obstruction. In only five of our patients was gastric mucin taken with little complaint. Eleven patients refused to continue taking the preparation after their initial discomfort had become ameliorated by mucin, diet or general care.

The exhibition of gastric mucin in gelatin capsules proved unsuccessful. The mucin escaped slowly from the capsules and instead of diffusing throughout the stomach contents to act as a "mechanical protectant" or to "buffer" free HCl, it formed tough, slimy "clots." These left the stomach slowly: we have washed them, undisintegrated, from a stomach as long as six and one-half hours following their being swallowed, where no obstructive lesion was present. In 5 instances, mucin capsules, only partly broken down, were recovered from stools as long as thirty hours after being taken per os. Finally, when gastric mucin is given in capsules, in order to exhibit an average dose of 100 gm. a day, an awe-inspiring number of large capsules must be prepared. Gastric mucin is not a cheap preparation, whether put up in capsules or taken from bulk. We abandoned the capsule as a vehicle of administration altogether after a relatively brief trial.

Our experience with Fogelson's gastric mucin as an aid in non-surgical ulcer therapy dates back approximately nine months. At first we employed a preparation supplied by Doctor Fogelson, but later ordered it in bulk by prescription. In all instances, we endeavored to exhibit the preparation according to printed instructions given by Doctor Fogelson to his patients at The Northwestern University Clinic.

CLINICAL EXPERIENCE

For purposes of comparison of our results with those of others, when possible (except in the presence of gross hemorrhage or threatened perforation) in this group of patients, we omitted the customary initial period of total abstinence from mouth-feeding or medication per os which is a characteristic feature of the "physiologic rest" method of treatment. The smooth, preponderatingly carbohydrate diet (outlined by us a decade ago), with small food volumes given, generally warm and frequently, was exhibited. Except in emergencies, medication other than mucin was omitted. It is to be emphasized, as C. Elliott[21] has done, that in their mucin management, Fogelson, Atkinson, C. Brown and others have consistently fed liquid or soft smooth diets and carried out other generally accepted therapeutic procedures while they subjected patients to gastric mucin therapy. Since such has been their routine and since it is well known that peptic ulcers may heal readily when diet alone is controlled, the precise evaluation of the clinical worth of administered gastric mucin practically has been difficult.

MATERIAL

Thirty-nine patients were studied (Table 1). There were no "acute" perforative ulcers; all were "obstinate" (histologically and chronologically) and chronic. No patient was experiencing his first "ulcer management": all had been "cured" from one to as many as 14 times, the average number of "cures" being 4. The average ulcer-type dyspeptic period had been 9+ years, with a minimum of 2½ years and a maximum of 33 years. Recrudescences had averaged, for the group, 8, with a minimum of 3 and a maximum stated as "innumerable." The average quiescent period between ulcer "attacks" was 5¾ months when not under active management supervised by physicians; then it increased to approximately 9½ months. Thirty-one of our patients were adult males, the remaining 8 were females. The age averaged 41 plus years, the range being 20 to 74 years. All were of the white race, approximately 80 per cent being American-born; the others were of foreign

Table I

TA UPON PATIENTS PRIOR TO PRESENT TREATMENT

(a) Unoperated

Ulcer location and type	
Duodenal ulcer, uncomplicated	12
Duodenal ulcer with protected perforation	5
Duodenal ulcer with partial obstruction	2
Gastric ulcer, uncomplicated	4
Gastric ulcer with hour-glass deformity	1
Gastric ulcer, prepyloric with partial obstruction	4
Ulcerated gastric polyposis	1
Total	29

(b) Patients with Prior Operations

Ulcer location and type	
Plastic surgery (at pylorus) for ulcer	3
Gastro-enterostomy followed by gastro jejunal ulcer	5
Partial resection followed by ulcer at anastomosis	1
Gastro-enterostomy and recrudescence of old ulcer	1
Total	10
Grand total	39

. *Miscellaneous Data*

Massive hemorrhage in	7
Average duration of entire ulcer history	9+ years
Patients having had previous "cures"	39
Surgery in addition to medical "cure"	10
Average number of non-surgical "cures" per patient	4
Average recrudescence interval when not supervised by physician	5¾ months
Average recrudescence interval under supervision	9½ months
Previous hospitalization (37 patients) averaged during "cures"	6+ weeks

4. *Age and Sex*

Female patients	8	
Male patients	31	
Average age: females	44	years
males	39	years
whole group	41+	years

5. *Roentgen Study*

Duodenal ulcer	Positive signs	26
	Inferential evidence	3
Gastric ulcer	Positive signs	9
	Inferential evidence	1
Gastrojejunal ulcer	Positive signs	3
	Inferential evidence	1
	History plus localized tenderness or fixation	1
Pyloroplasty: Positive signs in		3
Gastric polyposis: ulcerated multiple deformity		1
Partial gastric resection plus anastomosis		1

birth or extraction. The majority of our patients was in relatively easy, middle-class circumstances from substantial homes; none was destitute or of the usual dispensary class. Except when special observations were desired, test-meal data were secured after the exhibition of full mixed-meals. For more than seven years, in my clinic, no Ewald-type meals have been employed chiefly because I have not considered such a preponderatingly *carbohydrate* meal of small volume an adequate test for *peptic* digestion or for gastric motility.

Of the group, 10 patients had been subjected to one or more operations for the relief of duodenal or of gastric ulcer. Seven (17+ per cent) had experienced massive hemorrhage; 4 were bleeding when they came under treatment.

Of the 39 patients, 27 (70 per cent) were hospitalized or were bed-patients in their homes. Instead of the relatively high protein, acid-stimulating diet of Sippy, liquid and, later, soft and smooth, high carbohydrate, non-acid-stimulating, small frequent meals were given. We can find no reason for feeding food demanding acid and pepsin for its digestion should the object of diet in peptic ulcer be the control of free HCl.

Twelve patients (30— per cent) were ambulatory; either they did not feel sufficiently ill to take treatment in bed or, economically, they could not afford to stop work. None of this group had experienced massive hemorrhage.

RESULTS OF TREATMENT WHEN MUCIN IS EXHIBITED

Table II gives essential data relative to treatment and results and compares or contrasts the effects of ulcer treatment with mucin with those following the v. Leube-Sippy treatment and our regime of "physiologic rest."

Upon reviewing (Table II) the condensed summary of our experience, clinically, with mucin and upon comparing the results of mucin management with published records

of peptic ulcer behavior under other regimens, one is forced to conclude that the mucin treatment leaves much to be desired. Certainly, at our hands, this mode of therapy has not lived up to the expectations which had developed from the reports of Fogelson and of C. Brown.

It is granted that our experience has been brief and only a small group of patients has been treated, but these handicaps can be discounted since all the published work of the mucin proponants emphasizes the shortness of time requisite for the preparation to produce striking relief.

"Toxic" effects of gastric mucin have not been mentioned by Fogelson, Atkinson and C. Brown; in fact, it has been stated that gastric mucin caused no untoward signs or symptoms. Osgood[20] has stated that toxic symptoms may occur. Our experience has not supported this. Cases No. 9882 (gastrojejunal ulcer), 4934 (recurrent peptic-gastric ulcer with old gastroenterostomy), 9748 (chronic duodenal ulcer), 10,098 (lesser curvature, chronic ulcer), and 10,215 (ulcerated gastric polyposis) experienced definite ill-effects following the exhibition of 120 gm. of mucin daily. The disability appeared within an average time of three days (range, sixteen hours to five and one-half days). The patient felt weak ("all in"), and "queer," was listless but irritable and complaining on attempts to talk with him or carry out daily care; had light-headedness and slight mental confusion; sometimes vision was "blurred"; the face and body skin appeared pale and was cold and usually moist. Pulse was of low volume and rather frequent; the temperature at first tended to be subnormal but with ten to twelve hours increased 1 to 2°F. The blood pressure fell 10 to 25 points (mercury manometer). Nausea was a pronounced symptom and several patients vomited, 2 to the point of exhaustion and dehydration. Attempts to continue mucin administration aggravated the condition so that, in 3 patients, general stimulation became necessary (caffein sodium-benzoate, hypodermatically, external heat and rectal feeding). Disability lasted several days. Gradually, as mucin was omitted from the management, the condition was restored to that prior to the appearance of toxic symptoms and signs. In character, this toxic phase of gastric mucin treatment resembles what follows in patients who have passed through an acute mild protein shock or are convalescing from an acute, severe "bilious" attack. Our knowledge of the channels through which gastric mucin becomes operative toxically is incomplete. We have had no adequate opportunity to study the blood chemistry or the urine during the toxic phase. One is rather inclined to the opinion that when gastric mucin is not well borne, there occurs deficient protopexic function of the liver, so that partly-split mucin or excess amines pass through into the circulating blood. What the effect of mucin upon the digestive efficiency of trypsin and of *succus entericus* is, it is not possible to state, though this should be considered. In such circumstances, where there is excess of amines there occurs resemblance to the status present in high intestinal obstruction.

The daily dosage of mucin was, with the limitations stated above, approximately 120 gm. For the first week of management, although not without much urging and diplomacy on the part of nurses, such dosage commonly was possible. Since it is during the first few days that improvement has been stated as being so startlingly striking, even when after a week patients rebelled and less mucin was exhibited, our results are comparable with those of other clinicians. Early, it became apparent that inasmuch as disappearance of free HCl bore but casual relationship to the quantity of mucin given and since no one has advanced work proving how much mucin is required to "control" the free HCl in health or disease, it appeared that dosage was largely empirical. In peptic ulcer, as is true also for the normal healthy adult, the total quantity of gastric juice secreted in twenty-four hours varies greatly.

Accuracy of dosage would appear to call for an observation-period on each patient during which his volume-output of gastric juice and incidentally his free HCl are calculated. Otherwise, the daily dosage is but guessed at, just as at present is the quantity of alkali administered in the v. Leube-Sippy management. If, as is believed, gastric mucin acts as an excessive histamine-like stimulus and produces a pepsin-poor (or absent) gastric juice, it is likely that revision downward of dosage will be required. At any rate, we feel that there remains much to be done before accuracy of dosage for the individual can be determined. On account of the possibility of "toxic" effects with mucin, it is our belief that excessive dose, with its dangers of large amine (or derivatives) accumulations in the upper jejunum should be avoided.

With our patients it is interesting to note that limitation of the supply of mucin to 20 to 30 gm. daily (in 2 patients,

TABLE II

COMPARISON OF RESULTS OF NON-SURGICAL MANAGEMENT OF PEPTIC ULCER

Data on Patients	Mucin Therapy[14] (Author and Atkinson)	R Brown's[4] Data for v Leube-Sippy Management (Continuous Alkalization)	"Physiologic Rest" Regime (Author[5])
Average days in hospital	4 3 wks (Atkinson 3 1 wks 8 cases)	No statement follows treatment	3 7 wks
Days constantly in bed	13 days (Atkinson, Fogelson not stated)	No statement follows treatment	9 days
Pain controlled, average	6+ days (Atkinson, "2nd or 3rd day")	No statement follows treatment	40 per cent in 24 hrs 81 per cent in 3 or less days
Bleeding stopped, average	7½ days (Atkinson, "no longer than 9 days")	No statement follows treatment	92 per cent within 5 days
Nausea stopped	5 days (Atkinson does not state)	No statement follows treatment	1 5 days
Vomiting stopped	3 to 4 days (Atkinson, "almost immediately")	No statement follows treatment	18 hrs
Abdominal tenderness absent	5 to 9 days (Atkinson, not stated)	No statement follows treatment	2 to 5 days
Blood improvement began	6 to 10 days (Atkinson, not stated)	No statement follows treatment	2 to 5 days
Symptom free	6 to 15 days (Atkinson, 1 7 days)	No statement follows treatment	96 per cent within 3¾ days
Acid control	Irregular in relation to clin picture (Atkinson same)	No statement follows treatment	1 to 4 days
Favorable roentgen films	18 cases (49 per cent) (Atkinson, 18 cases all improved)	No statement follows treatment	68 per cent
No change in roentgen films	15 cases (39 per cent) (Atkinson declares none)	No statement follows treatment	14 per cent
Unfavorable roentgen films	6 cases (15 per cent) (Atkinson. "none has larger niche or more spasm")	No statement follows treatment	18 per cent (stenoses large scar, etc)
Effect on time interval of recrudescences	Apparently to be lengthened (Atkinson does not state)	No statement follows treatment	From average interval of 9½ mos to average interval 32 mos.
Recrudescences	4 cases (Atkinson does not state)	"Approximately 20 per cent"	14 per cent
Present status	11 cases (29 per cent) still ill, 28 cases (71 per cent) quiescent but time interval too brief for final opinion (Atkinson noncommittal C Brown· "Improved after mucin for 1 year", 37 cases [15])	49 8 per cent "cured", 16 7 per cent "greatly improved", 10 per cent "improved"	Of 470 patients, 361 (77 per cent) cessation of ulcer process Of 66 patients coming to operation in 54 (82 per cent) healed scars demonstrated

10 gm.) allowed 9 patients to remain just as comfortable as previously when they had been taking several times this quantity. Such observation would tend to support my opinion that if gastric mucin exerts any beneficial effect it is due more to controlling *peptic* than *acid* "corrosion." With regard the "protective" effect of gastric mucin, it is sufficient to say that no one has shown experimentally, at laparotomy or autopsy how much gastric mucin is required to "coat" an ulcer base to protect it from proteolysis or that mucin "coating" *ever* occurs.

While *the results of treatment* are detailed in Table II, several observations require emphasis. Under mucin therapy, our hospital patients required incarceration considerably longer than is stated by Atkinson as his experience and slightly longer than when managed by my "physiologic rest" regime. It is unfortunate that from neither the reports of the late Doctor Sippy and of his colleague R. Brown, statements are made regarding how long treatment lasts or how the patients fare while undergoing "continuous alkalinization" management. They give only data respecting end-results. Upon mucin exhibition, Table II shows that the patients averaged longer in actual bed rest, pain demanded a longer interval for its control, vomiting, bleeding and abdominal pain persisted longer and blood improvement began later than when our patients were on our routine treatment. While Atkinson's statistics in some respects are more favorable than those I present, yet on analysis it will be seen that the general trend of his experience with mucin is in line with ours. Neither his nor my mucin data would seem to be an improvement upon those accumulated from ulcer patients subjected to the "physiologic rest" regime. The responses to the "acid test" of Palmer,[22] or to Hardy's[23] improvement upon it, similarly are confusingly variable. Certainly such responses would not appear to indicate that, if gastric mucin, administered per os, has the property of mechanically protecting injured visceral mucosae, such a characteristic has been exerted, while, though clinically comfortable, he retains positive Hardy phenomena.

Roentgen proof of the beneficial effects of mucin must be accepted only following careful scrutiny and comparison of films. From its inception, peptic "ulcer" is a *mural* lesion: the mucosal, ulcerative, localized defect is a complication of what, initially, was a non-ulcerative, mural focus of tissue damage. Even if one grants that gastric mucin, administered by mouth, mechanically protects ulcerated visceral linings, no one has shown that such surface protection influences favorably the mural defect. If roentgen films serve to visualize only ulcer craters, stenoses, localized or general abnormal muscular activity as a consequence of ulcer "irritation" arising at the mucosa, one must realize that though mucosal reepithelization occurs, much pathology still may remain in the visceral walls to remain capable of producing great disturbance in function. Indeed, the very *favorable effects of healing* of ulcer may result in scar which, roentgenographically, may be seen as added deformity, as stenosis, adhesion to neighborhood structures, delayed emptying-time or the hyperperistalsis associated with gastric contents having to negotiate stenoses. Hence, *greater* deformity, or peristaltic activity, may not be an *unfavorable* roentgen sign; often they are concomitants of healing. Absence of "niches" or of spasm must be interpreted carefully; not rarely, films vary much accordingly as exposures happen to be made with stomach, pylorus or duodenum in *systole or diastole*. Comparison of films exposed during each phase is needed to avoid erroneous conclusions. Following the hypodermatic administration of atropine (to the patient's individual point of tolerance, with a time interval sufficiently adequate) changes in contour, spasms, "stenosis" and visceral emptying rate must be judged comparatively. Rarely are the conditions which have been mentioned satisfied; hence, much confusion has re-

sulted from attempting to compare or contrast roentgen signs following ulcer therapy with mucin or other agents, signs which are in no wise of like foundation.

Atkinson states that in 18 patients the roentgen films of all showed improvement; the evidence of improvement he does not detail. In none of his 18 patients were unfavorable roentgenograms observed nor did any films represent conditions as they had been before the patients were treated by smooth diet and mucin. After employing the v. Leube-Sippy management, R. Brown reports no data concerning changes in roentgenograms. In our patients who received mucin, the films were what we considered favorable in 49 per cent, there were no changes in 39 per cent and in 15 per cent films revealed greater degrees of deformity, even though, clinically, the patients were comfortable. "Spontaneous healing" may account for marked changes in gastric contour; such often accompanies simple rest of the stomach as R. Brown demonstrated recently in an instance where the v. Leube-Sippy treatment had failed and he fed the patient by duodenal tube for a considerable period.

When our "physiologic rest" regime was employed, favorable roentgenographic films were available in 68 per cent, but in 18 per cent the films revealed greater than primary deformity as healing scar and its consequences had accumulated.

We feel that the evidences of improvement as revealed roentgenographically after mucin therapy by Fogelson and his group, and, certainly, in our own experience, do not warrant the drawing of supporting opinions.

Ulcer recrudescences following mucin therapy must be expected. We feel that not sufficient time has elapsed in our own group of patients to permit drawing worthwhile conclusions. It has been our custom to allow a two-year interval free of recrudescence before we feel that the mural lesion in the viscera is quiescent in a way which could be called "healed." Even then, we have had patients who had passed through years of short-interval recrudescences which seemingly, spontaneously stopped, have their affections again return dramatically as massive hemorrhage or perforative attacks, as long as five to thirteen years after the stormy, short-interval recrudescence phase. The status of affairs at the mural nidus, not at the mucosa, determines whether or not peptic ulcer will recur. Of 470 ulcer patients which we treated by our own regime, of those who had recrudescences, the symptom-and-sign-free intervals were increased from an average of nine and one-half months to thirty-two months. Fourteen per cent continued to have relapses. C. Brown's report that 37 patients treated by smooth diet and mucin had been "improved for one year" is encouraging.

Physical Well-being. Much has been said by Ivy for experimental animals, and Fogelson for patients, regarding the marked physical benefits and improved "nervous tone" of patients who were experiencing mucin therapy. Unfortunately, we have not observed these effects. However, we believe that when patients or experimental animals tolerate mucin well, physical benefits may follow. Mucin, as split in the jejunum, has rich potentialities as a concentrated food. The daily ingestion of 120 gm. of this glycoproteid serves as a strong "boost" to the limited ration given a dog which has been subjected to experimental surgery or a human to whom a milk, egg and cream diet is given. Potentially, if not actually, 120 gm. of mucin may mean an excess of 500 food calories. This "sugar protein," when cleavage has occurred, has a potential and doubtless actual nutritive value both from its "sugar" and its amino radicles. The latter are end-digestion products, quickly supplied and since they appear in the upper jejunum doubtless, within limits, are quickly absorbed.

In 1913,[24] clinically, I was able to show that by feeding directly into the duodenum such artificially prepared end-digestion

products as the amino acids, patients affected with grave degrees of malnutrition rapidly took on weight and experienced marked improvement in blood, strength and physical and mental well-being. In ulcer patients who are taking large quantities of mucin without difficulty and over long periods, the improved nutrition resulting therefrom might conduce to ulcer healing.

DISCUSSION AND SUMMARY

Peptic "ulcer," esophageal, gastric or duodenal in location, rarely begins as a *mucosal* lesion, even when appearing as the classic, acute, "punched-out," perforative type, graphically described by Cruveilhier. The primary visceral defect and the focus from which arise recrudescences, are a *mural* disturbance in tissue-architecture and specific, secretary function. *Mucosal* damage, which converts the *mural* lesion into one of *ulcer type*, is a complication or extension of *mural* damage demanding treatment, because then the *mural* lesion is limited in its capacity to repair by the proteolytic action of gastric juice. When the mural lesion has reached the stage in its pathologic history when it has assumed ulcer form, extension of mural damage continues until mucosal epithelization is reestablished; such epithelization may take place with restoration of normal secretary structures or without.

"Ulcer" healing, therefore, is resolved into two distinct and definite phases: (1) epithelial reconstruction, partial, without normal, histologic, glandular architecture or with such, and (2) the disappearance of the primary *mural* lesion. Unless the original *mural* damage is repaired by scar sufficiently adequate to replace the entire localized pathological condition the *mural* fault persists and hence the *mucosal* destruction, which consequently exposes the mural lesion to proteolysis, again may appear. In such circumstances, pathologically and clinically, it is stated that the peptic ulcer has "recurred."

There would seem to be many local, systemic or constitutional disharmonies capable of producing visceral *mural* pathology which may extend so as to include the *mucous* membrane and hence convert the *mural* lesion into one of ulcer-form.

It is evident that the therapeusis of peptic ulcer calls for *first*, the eradication of the *mural* lesion by excision or by measures which allow the development of scar tissue thus accomplishing the same purpose, and *second*, the attempt to restore mucosal epithelization, functionally differentiated or not. *Both* mucosal and mural healing are requisite for pathological and clinical cure: only by the healing of the mural lesion can recrudescences be avoided.

Non-surgical management of peptic ulcer appears to be followed by a form of healing, sometimes permanent, in from 60 to 77 per cent of instances. When management is directed toward placing the mural fault in conditions most favorable to functional rest, healing appears to occur more rapidly and to be more permanent (82 per cent) than where the management is directed chiefly toward *mucosal* restoration by measures (medicinal or otherwise) which so alter visceral contents as to prevent mucosal (and, secondarily, mural) proteolysis.

Fogelson's gastric mucin has been advanced as an agent which prevents mucosal proteolysis by "buffering" free HCl, by mechanically protecting injured mucous membrane and, perhaps, by improving systemic capacity for healing through the agency of increased nourishment. Proof seems to have been adduced by Ivy, Fauley, Kim and Whitlow that, in certain circumstances, gastric mucin prevents pathologic proteolysis of visceral tissue. Fogelson and others believe that, clinically, in humans, evidence is available which substantiates the work of those who have worked with experimental animals.

In a study of 39 patients affected with proved peptic ulcers, we have not been able fully to substantiate the claims of Fogelson, Atkinson or C. G. Brown or to demonstrate that mucin therapy adds factors of value when exhibited in connec-

tion with a suitable dietetic, hygienic and constitutional regimen, unless some worth may lie in a form of superalimentation by mucin as a food. As would be expected, since the *mural* lesion of ulcer, essentially, is not controlled by mucin therapy, recrudescenses are possible. In our experience, management by Fogelson's gastric mucin has proved unsatisfactory. Not only has it brought no specific clinical benefits not secured by other means, but treatment is handicapped because gastric mucin is difficult to secure and to preserve, is obnoxious as to odor and taste, is difficult to administer *as is* or with food, is "rule of thumb" as to dosage and effects, is expensive, leads to a false sense of security on the part of patients, and, in a group of patients observed by us, appeared to have certain toxic potentialities.

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THE STONELESS GALL BLADDER

A STUDY OF OPERATIVE CASES*

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EXPERIENCE has demonstrated that stoneless gall bladders are found in approximately one-third of all operations undertaken for the relief of symptoms diagnosed as of gall-bladder origin. As they are encountered at the operating table, these stoneless gall bladders present problems not found in gallstone cases. When stones are present, the surgeon can proceed at once with absolute confidence based on the stones themselves. On the other hand, the stoneless gall bladders more often than not present very little evidences of unquestionable gross pathology. If the surgeon is to proceed with confidence and good judgment in these cases, his decisions must be based on definite knowledge applicable to this particular class of cases.

Out of 433 operations on the gall bladder performed by me up to July 1, 1931 stones were absent in 113 cases. This series of 113 stoneless gall bladders will serve as the text for this discussion. The relatively low incidence of only 26 per cent of non-calculous cases in my series may be due simply to a statistical coincidence or it may be that it reflects the results of a certain degree of skepticism on my part concerning the necessity of surgery in many of these patients without stones. Generally speaking, it may be said that stones are absent in about one-third of all operations performed under the diagnosis of gall-bladder disease. A great majority of surgeons report a fairly constant ratio between the stone and the non-stone cases with from 30 to 35 per cent classified as cholecystitis without stones. Occasional enthusiasts have reported as high as 60 per cent of their gall-bladder operations on non-stone cases.

One of the first difficulties encountered in the study of the surgical aspects of the stoneless gall bladder is the necessity of eliminating cases operated upon under mistaken diagnoses. In the very nature of things, the group without gallstones must contain practically all of the gall bladders erroneously removed. If the preoperative selection of symptom-producing gall bladder disease is 90 per cent accurate, the non-stone group will probably contain close to 30 per cent of erroneous diagnoses which must be eliminated before accurate conclusions can be drawn from experiences based on these cases.

Until pathologists learn to recognize pathological changes which can be constantly associated with preoperative symptoms and postoperative end-results, we must I believe depend on the postoperative history as the only reliable means of confirming a causal relationship between the preoperative morbidity and the gall bladder.

Much of the literature on cholecystitis has been compiled from a viewpoint quite different from the strict end-result test method adopted for this study. An extreme example of this other viewpoint is seen in the article of Jones and Palmer.[1] Based largely on *x*-ray and pathological data, these authors claim 97.1 per cent correctness in their diagnoses and yet they report a postoperative continuance of preoperative symptoms in 78 per cent of their cholecystectomized patients.

Of the 113 stoneless gall bladder cases operated upon by me, 21 are found to have

[1] *Am. J. M. Sc.* October, 1930.

* Read at the 44th annual meeting of the American Association of Obstetricians, Gynecologists and Abdominal Surgeons, White Sulphur Springs, W. Va., Sept. 14–16, 1931.

been operated upon too recently for end-result determination, or were traced less than six months after operation. For the purposes of this paper, these cases will not receive further consideration. Two patients died following operation, one of acute cholangeitis, and the other patient, an acute case with perforation of the gall bladder, died of bile absorption and peritonitis.

This leaves, for end-result analysis, a group of 90 operated cases traced for a total period of 312.8 years, or an average of 3.5 years for each patient. If I were to apply the ordinary space basis method of tabulating my end-results, I could say that as these 90 patients existed (in space) at the time when their conditions were last known, 67 were in average health and quite free from any symptoms for which their gall bladders were operated upon. Thus, using the statistical method ordinarily used by surgeons in reporting their end-results, I could say that 67 out of 90, or 74.5 per cent, of my stoneless gall bladder patients made complete recoveries following their operations. The trouble with the space basis method of reporting end-results is that in an investigation of this kind even patients operated upon under gross errors of diagnosis, as for instance acute pericarditis, diaphragmatic pleurisy, or what not, may all appear as cured at the time of the investigation.

If the 67 ultimately cured patients are re-analyzed on a time basis, it is quite evident that 19 out of the 67 recoveries cannot be definitely credited to the operation. Some of them were in patients known to have been operated upon under mistaken diagnoses, as for instance, acute pericarditis or partial intestinal obstruction. In others, the symptoms continued for months or years after operation and finally subsided spontaneously or after treatment in no way directed to the biliary system. If we subtract these 19 cases from the 67, we have left 48 cases, or 53 per cent, of the traced patients definitely cured by the removal of the gall bladder, and 42 cases, or 47 per cent, in which the operation had no directly demonstrable effect on the morbidities complained of. Of these 42 patients not definitely relieved, the gall bladders were removed in 19 cases because adhesions between the gall bladder and the surrounding organs were thought to be of a pathologic nature and might very probably account for the abdominal disturbances complained of. It is worthy of note that in only one of the 19 cases is there recorded a history of acute abdominal pains diagnosed clinically as gall-bladder colic. This patient had had only one of these attacks in the course of a long history of somewhat ulcer-like indigestion. Severe financial worries may have been the cause of his indigestion. The removal of the gall bladder had no really demonstrable influence on the subsequent history of any of these 19 patients although several of them recovered from or at least changed their complaints months or years after their gall bladders were removed. Very probably, some surgeons would say that the removal of the rather hypothetical focus of infection in the gall bladder was responsible for the occasional ultimate recovery. This hypothesis is not subject to proof one way or another.

Four typical strawberry-type gall bladders were removed from patients suffering from supposed biliary types of indigestion with no demonstrable effect on their symptoms. None of the 4 gave a history of typical gall-bladder colic.

In 19 patients the subsequent history has definitely demonstrated that the gall bladder was not responsible for the symptoms complained of at the time of operation. In this group of known diagnostic errors, there are 7 heart cases, 2 of which were acute pericarditis, one, angina pectoris, one paroxysmal tachycardia, 2 myocarditis and one goiter heart. In view of the great frequency with which cardiac disease may somewhat simulate gall-bladder symptoms, and the fact that many true gallstone cases demand operation irrespective of co-existing heart trouble, it is interesting to

find in this series only one case of the angina type of heart disease. Operation in the one case of paroxysmal tachycardia was based on a misleading history, the diagnosis being perfectly clear with the next attack. The two heart cases which proved most confusing and embarrassing were 2 cases of acute pericarditis. I missed both of these diagnoses absolutely although both patients subsequently had typical attacks of acute pericarditis with effusion.

Two patients after operation were found to be suffering from hysteria and not gall-bladder disease. Until we develop some pathognomonic, hysteria-proof sign for gall-bladder disease, abdominal surgeons will probably continue to be fooled now and then by patients suffering from this condition. The woman with hysteria and a fair knowledge of the symptomatology of gall-bladder attacks is a dangerous diagnostic possibility.

One patient, uncured by her gall-bladder operation, subsequently developed cancer in the region of the head of the pancreas. The beginning but not-yet-recognizable cancer was probably responsible for the symptoms complained of previous to operation. Two patients had syphilis of the liver which was provisionally recognized at operation. Both were cured by anti-syphilitic treatment. Three patients were found to be suffering from partial intestinal obstruction in no way related to the gall bladder or the gall-bladder operation. The subsequent histories in 3 other cases showed that ulcers of the stomach or duodenum were responsible for their symptoms.

In the general literature on the subject, I have found no statistically usable data exactly comparable to the experiences just related. Some surgeons seem to have made no diagnostic errors. Others rely entirely on the minute pathological data. Some pathologists have reported series of gall bladders examined running into the thousands with less than 1 per cent of normal gall bladders removed at operation. Such apparent accuracy in the selection of gall bladders to be removed either places the data in the "believe it or not" category or proves quite conclusively that the minute microscopical findings of the pathologist may or may not be related to symptoms.

After having related in some detail the failures incident to pioneering in this unsolved field of surgery, it is with some satisfaction that we turn to a group of 48 patients definitely relieved of their symptoms by the operations performed on their gall bladders.

This group of 48 cured cases comprises 41 cholecystectomies, 6 cholecystostomies and one cholecystenterostomy, the latter performed in the presence of a common duct stricture. I was rather surprised to find 6 cholecystostomies in this group. I believe that cholecystectomy is the operation of choice in these stoneless cases and have performed no cholecystostomies in stoneless cases in recent years. However, to show that cholecystostomy may give brilliant results even in the presence of severe pathology, let me cite the following case:

Mrs. V., aged seventy-three, operated on July 29, 1913 gave a history of gall-bladder colics extending over a period of ten years, with a particularly severe attack three weeks before operation, that had made her an invalid up to the time of operation. Operation revealed an acute empyema of the gall bladder without stones. Notwithstanding the evident infection present and the greatly thickened walls, plastered over with omentum, a simple cholecystostomy was done. This woman had no recurrence of colic or symptoms referable to her gall bladder until the time of her death in November, 1927, at age eighty-seven, fourteen years after her operation.

This group of 48 cured cases is composed entirely of cases in which there is apparently a very definite one-to-one relationship between cause and effect, namely, the operation on the gall bladder and the cessation of symptoms for which the oper-

ation was performed. Nevertheless, a study of the gross and microscopic pathologic findings embraced in this group fails to show any constant pathologic findings. The descriptions of the gross pathology range all the way from "gall bladder apparently normal but removed because of the clinical history of gall-bladder colics" to "acute purulent cholecystitis." Likewise, as regards the microscopic findings, the laboratory reported at least 14 per cent of these gall bladders as histologically normal. Slight chronic cholecystitis was the usual microscopic finding. One very striking fact is that so-called strawberry gall bladders or cholesterosis were described only twice in the 48 cured-by-operation cases; whereas 4 of the 23 cases in the uncured-by-operation group were described as typical strawberry gall bladders. As cholesterosis is said to be demonstrable in over 30 per cent of miscellaneous autopsies performed on adults, it is questionable whether cholesterosis per se is a symptom-producing condition.

It is only when we turn to the clinical symptoms that we find what I believe is a real clue by which we can recognize before operation the cases destined to be post-operative successes and those destined to continue with complaints much like those described before their gall bladders were removed.

Forty-two of the 48 cured patients described attacks of abdominal pain which were so characteristic that when the histories were transposed to statistical cards, they were credited as having had definite gall-bladder colics. Of the 6 cases not so credited, 4 gave a history of painful abdominal attacks which probably should have been classified as typical gall-bladder colics. One patient complained only of dull pains in the right upper quadrant. In another case, the chief complaint was described simply as gas in the epigastric region.

Gall-bladder-like colics are described in only 8 histories of the 42 patients not definitely relieved by their operations and in 4 of these 8 cases, the subsequent histories and findings proved the supposed gall-bladder colics to have been due to other causes.

The foregoing observations, as to both the pathologic conditions and the symptomatology, are in accord with results reported from the Mayo Clinic. In regard to the symptomatology, Judd says of the non-calculous cases: "If the patient had had attacks of severe, colicky pain he was almost sure to obtain relief by cholecystectomy, but the results of operating for indefinite symptoms were very unsatisfactory." This relationship between true gall-bladder colics and good end-results is apparently very definitely shown in my series.

Some surgeons in reporting the end-results of mixed stone and non-stone series have stated that their patients were relieved not only of colics but of non-colicky digestive disturbances in almost equal proportions. This has not been my experience in either the calculous or the non-calculous cases. In the calculous cases, I have relieved the colics and the sudden gas attacks so common in this condition and probably produced by lesser grades of the same mechanism that produces the truly painful attacks. Also, I have relieved the complications such as are caused by duct obstructions and active infections. In the non-calculous cases, I have relieved those patients who presented clear-cut clinical pictures of biliary colic. In both the stone and the non-stone cases, I have very largely failed to relieve those less definite complexes of indigestion which I have, in recent years, come to look upon as due to causes not associated with the gall bladder. Stated in another way, I would say that in my experience my late end-results have borne a direct relationship to severity and classicalness of the gall-bladder colics suffered before operation. When I have attempted to cure by cholecystectomy indigestions which I hoped were of gall-bladder origin but which were not accompanied by quite recognizable attacks

of gall-bladder colic, I have almost uniformly failed to get satisfactory results.

The authors of a recent treatise on diseases of the gall bladder and bile ducts state that in their opinion there is no more interesting chapter in the history of medicine than the gradual development of the idea that this organ is accountable for much of the dyspepsia, much of the partial invalidism and even a fair proportion of the cancer of the world. They also state: "This is in striking contrast to the older view which regarded as cases of chronic cholecystitis only those in which there were recurrent attacks of biliary colic."

This so-called newer concept of the morbidities due to cholecystitis may be correct but I doubt it. If the gall bladder were really responsible for the symptoms ascribed to cholecystitis without biliary colic, the removal of the gall bladder should promptly and permanently do away with the symptoms. In my experience this has not been the case unless biliary colics were the dominating feature of the clinical picture.

In conclusion, let me emphasize the following points:

1. All actual objective data that I have studied points to the conclusion that in the present state of our knowledge the one reliable indication of gall-bladder disease of a type yielding to surgical intervention is the presence of well-defined attacks of gall-bladder colic. If the surgeon is definitely certain of his ground relative to the clinical history of biliary colic, he can proceed to remove the gall bladder irrespective of obvious pathology recognizable at the operating table.

2. Just so far as the clinical picture of true biliary colics becomes doubtful, or the clinical picture shades over into ill-defined digestive disturbances of the general type that many surgeons have been wont to call the symptoms of the precalculous stage of cholecystitis, do the end-results of operative treatment begin to fail.

3. It seems highly probable that the indications for operation must be based and justified almost wholly on the symptomatology, and that little reliance can be placed on the pathologist's report until such time as pathologists learn to recognize a pathologic condition corresponding to the symptomatology.

TOTAL PERINEAL PROSTATECTOMY FOR THE SMALL PROSTATE*

A SECOND REPORT

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BOSTON

ON a former occasion I presented before this Society what I preferred to call an experimental group of small prostatic gland cases treated by radical prostatic surgery. For want of a better term I referred to these small glands in contrast to enlarged benign hypertrophics as fibrosis of the prostate —fibrous prostates.

Dr. Morrissey expressed what he felt to be his need of definition—and which still remains an important weakness in terminology for us all—when he asked me in discussion what I meant by the term fibrous prostate. My answer indicated that I had used the term to refer to those small glands in which the fibrous and muscular elements of their composition predominated and in which the glandular hyperplasia was either absent or limited in amount. Perhaps limited glandular hyperplasia should be considered the result of fibrosis because the enlarging glandular elements are limited in growth by the surrounding masses of fibrous tissue. There seems to be general agreement concerning the nature of the adenomatous hypertrophy type of gland. Few of us will disagree with the most excellent demonstration in Randall's recent book on pathology of the prostate. However, there seems to be many widely divergent opinions held, if surgical measures employed for relief are evidence, concerning the obstructing mechanism present in the small glands. One occasionally sees, in almost any clinic, operators either by the perineal route or suprapubically attempting to enucleate adenomatous tissue from a type of gland which when seen at autopsy or in specimens removed by the radical operation show little if any enucleable adenomata. The results of such surgery are not satisfactory to the urologist as a rule. Urology should be capable of producing surgically sound operative procedures applicable to the small glands and capable of producing results comparable with those for the benign hypertrophies.

Without doubt the present enthusiasm for punch operations had its beginning in attempts to relieve obstruction in these atrophic fibrous glands. They mean prostatic bar alone to many of us now. Yet we are all conscious of considerable variations in these glands. Occasionally one sees a prostate of atrophic nature associated with undeniable evidence of obstruction to the urinary flow which when removed in its entirety weighs but 4 or 5 gm. This gland has clearly caused total obstruction. There is undeniably either the glandular or fibrous bar type of obstruction associated with the small type of gland where the prostatic urethra appears cystoscopically to be freely open, and a urethrogram shows it to be so. On this type of case I do a cautery punch operation with the conviction that I am discharging my obligation to that patient both for the present and the future. There is a third type of small gland, which group I now believe to constitute a considerable portion of the whole where, even though the gland is composed chiefly of fibrous material, attempts at adenomatous hyperplasia has occurred in some portion of the gland. Small lateral lobes are held firmly approximated in the inelastic fibrous tissue

* Read at the meeting of the New York Society of the American Urological Association, October 28, 1931.

in such a way as to offer obstruction even after some relaxation of the gland is obtained by cautery treatment of the prostatic bar. In the most pronounced of these cases enucleation operation leaves behind much deformed prostatic tissue, often harboring infection, and yields the smallest of adenomatous specimens. Often the result functionally is excellent. In other instances the patient either remains functionally embarrassed or contracture from scar tissue requires long periods of dilatation. Adenomatous tissue occurs in such glands in any of the usual places such as lateral lobe, commissural lobes, subtrigonal nodules or miniature general hyperplasia.

It is as yet too early to determine what the fate of this latter type of gland will be in later life after treatment by median bar cautery alone. Already it seems to be true that infection remains a troublesome factor in some although the bladder has been enabled to function passably well after median bar relief. Carcinomatous degeneration is equally probable in the punched and the unoperated cases. The next ten years will show interesting end results from this surgery. Partial prostatectomy in the adenomatous hypertrophy as practiced a generation ago was abandoned as inadequate.

Meanwhile, I again offer for your consideration total removal of these glands except where prostatic bar alone exists. This I believe demands only section, and cautery punch is the preferred means for accomplishing it.

Since my first cases were presented, I have operated on a few cases by total prostatectomy, primarily for obstruction, but secondarily for extensive and long-standing prostatic gland sepsis. The operation has a definite application to this type of case. Hinman has recently reported 5 cases so treated.

I have already described the technique employed in performing total prostatectomy on the small gland, but will review it later. Essentially it is that employed by Young for total prostatectomy for cancer. When one operates for the small gland in contrast to the cancerous prostate, it offers no technical difficulties. In fact, I find that for me total prostatectomy for the small gland is less difficult technically than attempts at partial removal either by enucleation or dissection by either the suprapubic or perineal route.

To date I have performed this operation on 26 cases with a mortality of one. The patient who died had large bilateral diverticula and extensive renal damage. Death was due to bronchopneumonia on the ninth day after the prostatectomy. Diverticulectomy had been performed some weeks previous to the prostatectomy.

All living patients have been followed to date. Ultimate satisfactory results were obtained in all but one. This patient has continued to have leakage on exertion, coughing, or sudden motion. Even though sepsis was present previous to operation, the urine has cleared entirely. He voids in normal amounts.

One patient out of the 25 now living has already been reported in my previous communication as having had temporarily some degree of loss of control but recovered after four months. He is now entirely in control of his micturition.

One patient who showed much sepsis remained irritable for about a year. After the passage of sounds this symptom disappeared.

In a survey of postoperative results in prostatectomy cases which I have made during the past year both from the clinical aspects of restoration of satisfactory function and cystoscopic findings and by urethrography, total prostatectomy cases showed the most satisfactory results of all the types of operation which had been employed. From the clinical, cystoscopic and *x*-ray point of view, total prostatectomy gave us our best final results. Since some of our cases were operated upon several years ago, these results can reasonably be expected to represent final results.

CASES OPERATED ON	DATE
2	1924
3	1925
3	1926
1	1927
4	1928
3	1929
3	1930
7	1931
26	

The number of cases to which total prostatectomy is applicable will probably be but a small part of all the cases of prostatism. I know of but few statistics indicating the relative frequency of occurrence of the small gland. Randall's figures from autopsy cases are probably the fairest estimate of the relative frequency of hypertrophy to small gland types. While he chose to classify his cases into "gross pathological changes" and "median bar cases" perusal of his pages and reference to his photographs indicates that by "gross pathological changes" he referred to the large hypertrophied glands. All other types of obstruction except carcinoma, tuberculosis and abscess, he seems to have included in the second group. His figures are as follows: of 312 cases showing prostatism, 222 were due to "gross pathological changes," while in only 57 cases was the small type gland encountered. There seems to me to be great need for subdivision of this small group along the lines which I suggested earlier in this paper. By such subdivision suitable cases for punch and cautery procedures will be established as well as a group in which total prostatectomy will find its place as a useful addition to our armamentarium of surgical operations.

DISCUSSION

Dr. Edward L. Keyes: The Crabtree operation is not a suitable method, in my opinion, for attacking prostatic bars in young or middle aged men, since it almost invariably destroys the sexual function. But it does seem quite appropriate in the treatment of the truly sclerotic prostate, the type whose very existence Randall has denied. One of my patients died some years after an operation performed on him by me which had left him with about 100 to 150 c.c. of residual urine. Autopsy showed the bladder neck (where the bar had been bitten away) wide open, as at the time of operation, but the whole posterior urethra was just as sclerotic as ever, and perfectly rigid. I believe the retention of urine was due to the rigidity of the prostate. It is a practical illustration of what Dr. Crabtree means in stating that some patients will not be relieved by operation other than total prostatectomy. However, I confess such cases are less frequent in my experience than they seem to be in his.

I am at present engaged in reviewing this type of case, and have been interested to find that some of the cases have gone a great number of years quite satisfactorily after various operations attacking different types of prostates ranging from the simple bar to the totally sclerotic type due to prostatic abscess or other inflammation of the gland sufficient to render it rigid throughout.

I have operated upon cases by other forms of operation than Dr. Crabtree's and found they required a great deal of after-treatment. And I think some of his will require a fair amount. For after all, some scar tissue is left and even at times a crookedness of the urethra. In fact, he mentions a case done a year or so ago which shows some tortuosity of the posterior urethra needing to be ironed out.

Dr. Nathaniel P. Rathbun: When Dr. Crabtree made his first presentation here two years ago, I confess I was not at all enthusiastic about this operation. It seemed to me a logical procedure, but too radical. It appeared that in the hands of the average operator, it might have unfortunate sequelae, such as incontinence, that it would be difficult to do the operation without damaging the sphincters or causing enervation. Having seen Dr. Crabtree perform this operation, and having done it myself, and hearing again his further report on its satisfactory results, I have reversed my position. I am very enthusiastic about the Crabtree operation. I feel that he has made a major contribution to urology in his modification of Young's operation.

My experience in handling these cases in the past has not been satisfying. I have

had very few altogether good results. I have treated them in various ways, by punch, suprapubic prostatectomy, going in with the finger and fingernail and gouges to chew out pieces of tissue amounting in the aggregate to the size of a normal prostate, etc., altogether unsurgical procedures, I have felt. Residual urine has been reduced, but frequency has continued in many instances almost as bad as before operation.

I agree with Dr. Keyes (in fact, Dr. Crabtree says himself) that these cases for his procedure will have to be carefully selected. It seems to me that a year or two ago, before I was convinced of the value of Dr. Crabtree's operation, I saw many more opportunities to test it out, and that since I know its merits I have seen very few, in fact, the total number of cases coming to surgery of late has been decidedly less. I have performed this operation only once or twice lately. I was, however, impressed with the ease and facility with which it can be done. I do not claim to be an expert in surgery of the perineum. I have felt that the Young prostatectomy offered difficulties in my hands; and yet with my limited experience I felt at ease with Dr. Crabtree's procedure. It is, in my opinion, simpler than the ordinary form of Young prostatectomy. Dr. Crabtree has illustrated to us the way the bladder neck is peeled back; and the ease and facility of completing the anastomosis of the open ends is striking. Also, it is easy to get the feeling of a smooth, tight sphincter after removing the prostate.

I believe this operation may even be extended a bit. I have in mind another group with unsatisfactory results, the cases of prostatic calculi. Such cases as I have seen have always been associated with fibrosis of the prostate, many of an obstructive nature. They offer a trying experience at all ages. I have gone in from above and taken out pieces of prostate, and removed gravel and stones. And on sending the patient down for a roentgenogram before leaving the hospital I have been surprised to find that there were still calculi at the bladder neck. One of the cases in which I employed Dr. Crabtree's operation was in this group. Both the calculi and the prostate were removed, and there was an absolutely perfect recovery. There was no residual urine, the patient passed a good stream under perfect control, and slept all night.

Another group of cases in which, as one becomes more expert, this operation may be useful is the early type of carcinoma of the prostate, calling for operation of a radical nature. Most of the cases that I see in which the diagnosis is a certainty are beyond my skill for radical operation. Occasionally, however, we see cases in which early carcinoma is suspected but in which we cannot definitely make a diagnosis. A few years ago we hesitated to recommend a radical prostatectomy on such patients because we were afraid of incontinence following operation. I feel perfectly at ease on that point now and shall not hesitate to recommend and perform a total prostatectomy on just that type of case, feeling that I may effect a radical cure in an early case, or if I am mistaken in my diagnosis, that I have done the patient no harm.

Dr. Oswald S. Lowsley: I think that for one as proficient in this operation as Dr. Crabtree is, to have operated upon only 7 cases by this method in the past year also earns him the title of being the most modest pioneer I have ever met. It might prove an example to some of our other pioneers.

No discussion of prostatic surgery can omit the various punch operations, the Young operation, and such procedures, of course. I am sure these will all be tried on the type of prostate Dr. Crabtree has selected for his operation, and quite agree with him they will often not prove suitable procedures. For the fibrosed prostate such as he has described, total prostatectomy seems exactly the thing to do. Yet the cases which can be so treated are very limited indeed. Since hearing his paper two years ago I have been stimulated to try the operation, and have performed it twice. I have been searching very assiduously for cases in which I felt justified in doing it. I will admit, also, that one of these prostates was totally removed because I thought it an early case of carcinoma; and I still believe it to be such, in spite of negative reports on specimens. The patient is apparently well now, yet I feel the condition is a malignant one.

The question of technique taken up in Dr. Young's recent article is an important one. That is, it emphasizes the value of preserving the network of blood vessels on top of the prostate, and leaving a foundation layer of fibrous tissue and part of the capsule, if necessary. Otherwise there is a lot of bleeding

and injury to the nerves, which is of importance in connection with continence afterwards. If we destroy this entire tissue, incontinence is much more likely to follow.

Dr. A. R. Stevens: It seems to me that Dr. Crabtree has been very careful to confine this type of prostatectomy to a very small group of cases. The fact that he has been interested in the operation over a period of seven years and yet up to 1931 has found no more than 4 suitable cases in any one year, indicates a belief that there is only a comparatively small proportion of prostatics which should be handled in this way. That group may properly include certain cases of fibrosis which come after an obviously acute infection. But the group he has in mind, and in all probability the one we will encounter most often, is that of fibrosis in its comparatively broader meaning—i.e., with no history of acute infection of the prostate.

My difficulty has been to differentiate between the cases of prostatic obstruction, not due to carcinoma or hypertrophy, which will be helped by the punch operation and those which will require total removal of the prostate. I have operated in this way only twice, with this in mind: that the smaller operation should be tried first and prostatectomy employed on individuals who had had punch operations without relief. One case was an individual who had foul residual urine and multiple small diverticuli of the bladder (part of which contained calculi). On his first admission these calculi were removed, and a punch operation performed under suprapubic control. He was more comfortable following this, but still had frequency and foul urine, with more or less residual. He returned eight months later with increased frequency, purulent urine, one calculus in a diverticulum; and it seemed wise to operate to remove this calculus. Again a piece of the prostate was removed by punch operation. He healed after this, but still had about 2 ounces of residual urine and very marked frequency by day and night. We were at a loss to know what to do. A great many bladder irrigations were given, without relief. It occurred to me that by taking out the whole prostate according to Dr. Crabtree's method and getting rid of the residual urine, the formation of calculi would be less likely to take place, and at the same time the patient would be given a better opportunity to overcome infection. The result was that he was relieved of his residual urine, made an excellent recovery, had perfect urinary control; but when last heard from, about four months after leaving the hospital, he was still very uncomfortable and had marked frequency of urination, obviously due to the infection.

In one other case where the prostate was completely removed (a man fifty-two years of age with a history of twelve years of urinary frequency), within twenty-four hours after he entered the hospital and before we had had an opportunity to examine him thoroughly, he had a very marked discharge of pus from the urethra, quite apparently coming from a ruptured prostatic abscess. He was under treatment with hot rectal irrigations and the usual therapy indicated in such conditions for many weeks. He finally developed considerable fever, which was regarded as caused by infection of the prostate and bladder. A suprapubic opening was made. The fever came down, and afterwards we did a punch operation. He healed, but still had 10 ounces of residual urine. We therefore removed the prostate according to Dr. Crabtree's method; and while the first case mentioned offered no difficulties, this one was a more or less complicated one, inasmuch as he had a great deal of periprostatitis. He finally healed without too much difficulty, was relieved of his residual urine, had control when lying down, but on walking around there was occasional slight leakage.

Dr. J. Sturdivant Read: I agree that this procedure of Dr. Crabtree's is a distinct contribution to prostatic surgery. The selection of cases in which it is indicated has been discussed by the previous speakers, so that I will consider it from another angle.

First: Surgically, is it easily done in the average competent hands?

Second: Is there any particular shock to the old or feeble patient?

Third: Is it more apt than any other perineal procedure to be followed by permanent leakage of urine from either a perineal wound or from the meatus?

I can only give you the results of 4 cases done by myself; and as I am not very familiar with perineal surgery (most of my cases for years have been done suprapubically), I think that my conclusions are a fair answer to the foregoing questions.

CASE I. That of a feeble old man of seventy-three years, admitted in retention. Suprapubic drainage was necessary for thirty-eight days before the patient's general condition would allow of further surgery. We followed Dr. Crabtree's procedure. The usual curved incision was made, the urethra cut across, and the prostate delivered and resected. I was surprised by the amount of slack between the cut urethra and the bladder. It was easy with an ordinary needle holder and curved needle (no boomerang or gadgets) to place three chromic sutures approximating the bladder neck and the cut urethra. As this was my first case, I wondered if the approximation would hold, and feared a permanent fistula. The patient made an uneventful recovery in three weeks. After five months I find that he urinates without difficulty, has no residual urine, and has complete control. Forty minutes before operation, 2 c.c. of 0.5 per cent nupercain were given. Anesthesia was complete during operation and for two hours postoperative.

Cysto-urethrogram taken while voiding (Fig. 1) shows good approximation and normal appearance of the prostatic urethra after prostatectomy. The narrowed portion of the urethra between the bulb and the prostatic part is due to the action of the compressor urethrae muscle.

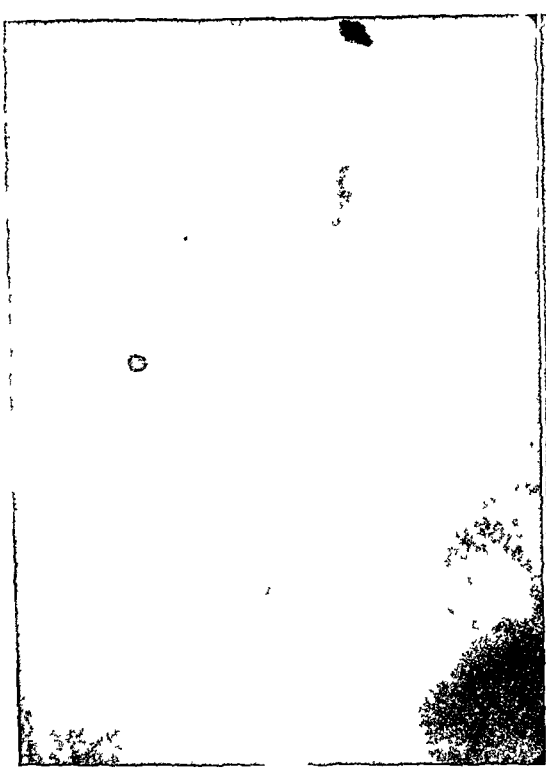


FIG. 1.

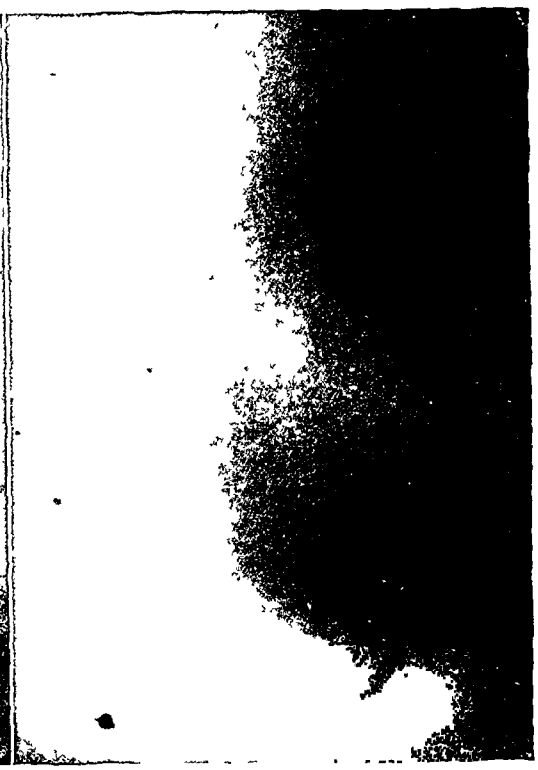


FIG. 2.

CASE II. Man sixty years old, with a small fibrous prostate. The patient was operated on in the same manner. In this case, hemorrhage necessitated packing in one corner. Consequently there was considerable infection. The night of operation, the patient pulled the urethral catheter out, and leakage of urine through the wound added to the infection. Two and one-half weeks afterwards the exposed urethra could be seen with urine trickling through. An urethral catheter was inserted, worn comfortably for two and one-half weeks, the skin flaps sutured, and complete healing took place. The result four months later was reported as disappearance of the 8 ounces of residual urine, no perineal fistula, and complete control. The pathological report was prostatic adenoma with focal malignant areas.

Cysto-urethrogram taken while voiding (faint outline because patient moved) shows smooth junction of bladder and urethra. (Fig 2.)

Case III. Adenocarcinoma of the prostate and vesicles. Symptoms of mild prostatism for four years, severe for three months. In retention for three days. Suprapubic drainage under spinal anesthesia, for ten days. Prostate was fixed, one lobe projecting into the bladder along the posterior surface of the internal sphincter. Bladder membrane healthy. Using Young' retractor, this prostatectomy proceeded smoothly. The vesicles were cut across at a convenient point, along with the urethra. A No. 20 Fr. urethral catheter was put in place. There was complete closure in seventeen days, and the patient was home in thirty days.

Clinic note five months later: Function perfect. Urinates every three hours. Cysto-urethrogram taken while voiding (Fig. 3) shows smooth junction of urethra and bladder, the ureter narrowed near the bulb, due probably to contraction of the compressor urethrae. The bladder is elongated, due probably to fixation in the surrounding tissues.

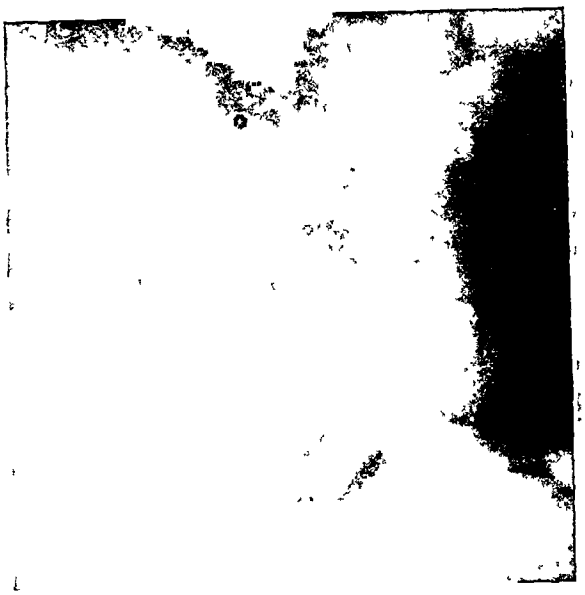


Fig. 3.

Case IV. Man sixty-six years old. Symptoms of prostatism for four years, frequency every two hours, and residual of 2 ounces. Prostate per rectum small; adenoma. Two c.c. of 0.5 per cent nupercain produced a 60 per cent successful anesthesia. The membrane in the bulbous urethra was extremely thin. The urethra was approximated to the bladder with three chromic sutures, which were tied loosely for fear of cutting through. Nevertheless, because of the large amount of slack there was firm union. There was a slight perineal fistula for thirty days, but four months later the clinic report is "no residual; frequency every four hours; control perfect."

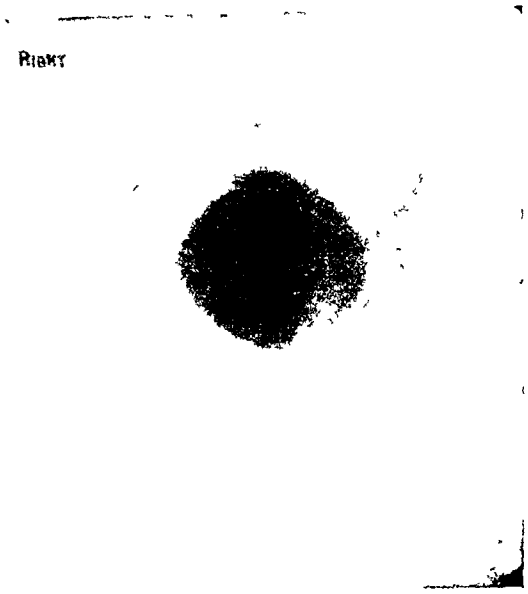


Fig. 4.

Cysto-urethrogram in semilateral position taken while voiding (Fig. 4) shows smooth junction of the urethra and bladder, and the usual narrowed membranous urethra, which is interpreted as action of the compressor urethrae muscle.

The question of when to use this operation is open to judgment. But I know that it is a simple procedure, that function in my 4 cases is perfect, and that there is no residual urine in any of these cases.

Dr. Edwin Beer: Of course the whole question resolves itself into just what cases are to be treated by Dr. Crabtree's operation. He has not outlined how to recognize them preoperatively. It seems to me rather difficult to determine without opening the patient up. In a case with fibrosis of the prostate and contracture of the neck of the bladder, one often detects, after wide incision into the fibrous prostate, a narrowing of the urethra beyond the reach of the finger. Such cases are those which Dr. Keyes has mentioned. Under these conditions, a total prostatectomy seems indicated; but how to recognize it preoperatively, I at present fail to see. When the bladder is once opened and we have such

a finding, we can take out practically all of the fibrosis from above with the electro-cutting needle, down to the compressor muscle.

I would like to know what Dr. Crabtree has found in cases coming to autopsy some months after the operation. Judging from Dr. Read's pictures, I should say that the bladder retracts from the compressor muscle region and a new posterior urethra forms. I do not believe that in these pictures the neck of the bladder was near the compressor muscle. I noticed in the course of the discussion that some of the gentlemen seemed to be under the impression that the sphincter at the neck of the bladder controls bladder function. I am very definitely of the opinion that after prostatectomy the internal sphincter fails to function in the great majority of cases, and that urinary continence is entirely due to the compressor muscle. Many studies of the posterior urethra after prostatectomy, both by urethroscopy and injection, have shown in innumerable cases that the internal sphincter function was borne by the compressor muscle, this muscle alone supporting the column of water in the bladder.

Dr. Howard S. Jeck: In line with what Dr. Beer has just said, I should like to know if Dr. Crabtree found any temporary incontinence after any of his operations.

Dr. E. Granville Crabtree (*Closing*): In answering the queries of various members, I confess I do not know to just what group of cases this operation will eventually be applied. Experience will have to determine that. As Dr. Lowsley has commented, I do not believe I have been over-enthusiastic in operating on 26 cases over a period of seven years. My enthusiasm has been tempered by the one point emphasized so many times here, namely, that there is difficulty in making an accurate diagnosis of the size and condition of the prostate. I have felt that certainty of diagnosis was not always possible by cystoscopy, by rectal examination and the other usual means, and have therefore attempted to employ urethrograms for that purpose. I shall later publish my experiences with urethrography, so that you may judge how far I have gotten with this method.

My impression is that you here have extended the application of an operation which I had confined to the small type of gland essentially fibrotic in type. You have reported its use in doubtful cases of carcinoma. I believe such cases belong to this group. Some of them were actual cases of carcinoma, some believed to be and found benign. Dr. Read has reported an ordinary-sized adenomatous prostate operated by this method. Dr. Hinman, in his last publication dealing with prostatics, reported 5 similar cases with infection, and showed pathological specimens of quite large benign hypertrophies operated in this way. Dr. Keyes has suggested still another type of gland to which it is applicable, one to which I have perhaps referred but not been definite about.

I feel that time alone will give us definite knowledge of just what group of cases will be suitable for this operation. I am absolutely convinced that the number will be small. Total perineal prostatectomy will not revolutionize prostatic surgery.

Several speakers have reported residual urine after the operation of prostatectomy. I would call your attention to the fact that in neither my cases nor those mentioned by other operators here has residual urine been reported, although tested for in all cases.

As to autopsy findings, there has been but one performed, and that nine days postoperatively. All other patients are living.

Dr. Jeck has mentioned postoperative incontinence. One patient previously reported completely recovered normal control, and has continued in this condition for two and one-half years, though for four months postoperatively he had slight incontinence, that is, a few drops on exertion.

RECURRENT BENIGN PROSTATIC HYPERTROPHY*

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NEW YORK CITY

THE operation of total prostatectomy has always carried with it the thought of a complete cure and the absence of recurrence. Since Freyer demonstrated his operation (prostatectomy) in 1901, the procedure, with slight modifications, has been carried out in thousands of cases. Most doctors and all patients had believed or had been told that the procedure was an absolute guarantee against further similar trouble. However, at this time we are gradually beginning to change our conception and realize that recurrent prostatic adenomas are more common than we had supposed and may even produce complete urinary obstruction again and develop to a size even larger than at the time of the first enucleation.[1-4]

The adenomatous prostate tends to hypertrophy with age and increases constantly in size. In occasional cases this growth is rapid but is usually slow and may even have remissions. There is no tendency to necrosis, ulceration or metastasis. The growth is painless and the symptoms produced are due to pressure, except when hemorrhage occurs. Adrion[5] and Loeschke[6] showed that the prostate consists of three types of glands: mucosal, submucosal and prostatic. Motz[7] and Perearneau[8] wrote that all hypertrophy of the prostate arises from the mucosal or submucosal glands. Tandler and Zuckerkandl[9] and others have confirmed this work. The enlargement always begins in the inner or mucosal glands and is always central while the hypertrophic processes very seldom begin in the prostatic glands proper, or outer glands. However, the latter may occur and occasionally one sees an isolated spheroid of hypertrophy there. This undoubtedly accounts for certain cases of recurrence after complete prostatectomy (Young[10]). As the hypertrophic masses grow the unaffected prostate becomes fibrotic.

The cause of recurrence of benign prostatic hypertrophy seems to be a disputed subject. Takahashi[11] advanced the idea that the prostatic capsule is not a true capsule but a tissue composed of flattened glands which take on an activity of regeneration of the gland structure when the pressure has been removed by prostatectomy. Hedinger[12] substantiated this view by finding distinct recurrence of glandular tissue in autopsy specimens two and four months after prostatectomy. Tandler and Zuckerkandl, Honegger[13] and Blum[14] also support this theory. Freudenberg,[15] first an advocate of this theory, later came to believe that the regeneration did not take place from the glandular tissue of the so-called capsule, but from small adenomatous nodules left at the time of the first operation. Jacoby[16] studied the glands of the prostatic urethra and differentiated them into mucous, submucous and prostatic. The submucous glands form the cervical group about the vesical neck and may give rise to the pathological middle lobe and are also located on the anterior or ventral wall of the urethra. These urethral prostatic glands are the sites of the hypertrophic centers and are continuous with the prostatic glands in the prostatic capsule. He believes these centers become more active after prostatectomy and permit expansion to the point of producing obstruction.

The literature to date contains only about 40 cases: Andre one, Bazy one,[17] Constantinescu one,[18] Fedoroff one, Hedinger 2, von Illyes 4,[19] Loumeau 2,[20] Lumpert one,[21] Takahashi one, Zuckerkandl one,

* Submitted for publication December 14, 1931.

von Grigoracis 3, Pauchet 5, Frank one, Nogues one, von Krois 2,[22] Honegger one, Gregora 3,[23] Young 3,[10] Lewis one,[24] Cunningham 3,[25] Le Fur 2,[26] Freudenberg 3.

Grigoracis reported 1 per cent in 300 cases of suprapubic prostatectomy in the clinic of Rafin, and Illyes 3 in 470 cases. Bazy[17] and others comment that the perineal route is not so favorable for exploration for remaining glandular tissue or nodules as the suprapubic and therefore is more likely to be followed by recurrence.

Most of the patients reported are over sixty years old, though there have been a few below this age level. Some patients have no symptoms of regrowth but reveal such on rectal or cystoscopic examination. It has also been suggested that most prostatics die before a sufficient time has elapsed since the first operation to permit regrowth. The recurrence usually produces symptoms after eight to ten years, and are similar to those in the unoperated group. The examination may reveal lateral or median lobes, though the former are more common and in many cases even larger than at the time of the first operation. There is usually no fibrosis about the adenomatous nodules, so that the enucleation is no more difficult than at the first operation.

My patients were sixty-two and sixty-one years old respectively.

Case 1. S. M. sixty-two years of age, had been operated upon by a competent surgeon in November, 1926. A two-stage complete prostatectomy was performed at that time. The tissue was reported a pure adenoma. In September, 1930, he developed gross hematuria and later urinary retention, necessitating catheterization. Examination at that time revealed a large recurrent adenomatous hypertrophy of the prostate, the size of a small lemon (2 × 1½ in.). Palliative treatment was instituted and the patient was well for about one year. In September, 1931, the same symptoms recurred, but the prostate had become much larger. Residual urine totaled 4 fluid ounces. Cystoscopy revealed a moderately trabeculated bladder with marked intravesically and intraurethrally intruding lateral lobes. Vocal cord action could be demonstrated. An operation was decided upon. A suprapubic two-stage prostatectomy was performed and an adenomatous prostate, the size of a small orange (3 × 2½ in.) was easily enucleated. No abnormal amount of fibrosis was noted. The patient made an uneventful recovery and left the hospital in five weeks, voiding per urethram. Pathological report: cystic adenomatous prostate.

Case 11. H. L., sixty-one years old, had a complete prostatectomy in September, 1929, by a competent urologist. The pathologist reported adenoma. In August, 1931, the patient consulted me, complaining of urinary frequency. Rectal examination revealed an adenomatous regrowth, the size of a walnut (1½ × 1 in.) and residual urine of two fluid ounces. Cystoscopy revealed a moderate intravesical intrusion of the lateral lobes. The patient was treated conservatively and improved symptomatically. At the present time no operation is necessary, but if the adenoma continues to grow, a complete enucleation may become necessary.

CONCLUSIONS

1. Prostatic adenomas do recur and may produce symptoms as severe as at the time of the first operation.

2. Recurrence may be due to nodules left at the time of the first operation or activity in regenerative centers in the prostatic capsule.

3. At the time of the first operation one must be extremely careful to remove the entire adenomatous prostate with nodules, etc.

4. Two cases are reported: one had recurrent symptoms with obstruction within four years and had a second prostatectomy after five years. The second has symptoms with palpable enlarged recurrent adenoma within two years.

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[For Remainder of References see p. 269.]

PROBABLE BILATERAL TUMOR OF THE CAROTID BODY

REPORT OF CASE*

GEORGE DE TARNOWSKY, M.D.

CHICAGO

NOT only are carotid body tumors of rare occurrence (143 cases up to the year 1930 were reviewed by Bevan and McCarthy) but, including ours, only thirty-two tumors of this type have been removed without ligation of any blood vessels. Still more rare are the reports of bilateral tumors of the carotid gland, in which our report belongs, although the hospital records of the first operation are not available.

CASE I. Cook County Hospital No. 1187130. A forty-four year old Italian workman, giving a fictitious name on admission, entered the hospital September 12, 1930 for treatment of a swelling in the right side of the neck which had been present for twenty-six years.

Previous History: Twenty-six years ago (1904), he stated that a similar tumor had been removed from the left side of the neck by the late Dr. Camillo Volini, at the German Hospital (now Grant), Chicago. The tumor, according to the patient's statement, was solid; the incision was made at the apex of the left sub-maxillary triangle, the growth removed and wound closed without drainage. No recurrence of the tumor occurred. During his stay in the hospital it was noted that the right side of the neck had begun to exhibit similar swelling. The patient left the hospital without further surgical intervention but returned later (1906), when he was treated by a Dr. Senn for syphilis.

Present History: The tumor has remained stationary as to size and location and is painless unless pressure is applied against it.

Examination: A well-nourished white male of forty-four years of age, not acutely ill. Temperature 98.6; Pulse 78; Respirations 18.

Head, negative.

Neck: No adenopathy; on the right side of the neck, in the submaxillary region, is a tense, smooth, fluctuating tumor about 1½ inches in diameter, movable laterally and not tender. A distinct impulse can be felt which gives one the impression of being expansile in nature but which may be transmitted impulse from the carotid arteries. No bruit is heard, but the heart tones are audible over the tumor. There is an old scar in the left submaxillary region.

Chest: Lungs and heart negative. Abdomen: negative. Genitalia, extremities, reflexes, normal. Wassermann-Kahn reactions, negative.

Working Diagnosis: (1) Branchial cyst. (2) Cyst of submaxillary gland. (3) Thyroglossal cyst. (4) Aneurysm of carotid artery.

Diagnostic review, September 16. Above findings noted and confirmed. There is a small-egg sized painless nodule on the anterior surface of the right sternomastoid. Impression: (1) Lymph gland, either tubercular or metastatic carcinoma; (2) bronchial cyst; (3) small hygroma.

In retrospect it seems strange that the true nature of the growth was not even suspected because, in the September, 1916 number of *International Abstract of Surgery* the writer had presented a rather exhaustive differential diagnosis of congenital malformations of the neck, making the statement that "a solid tumor of long standing, situated in the anterior triangle of the neck, at or near the level of the lower border of the thyroid cartilage, should be looked upon as a possible carotid body tumor and removed before it involves the surrounding tissues, particularly the carotid sheath."

Operation, September 17, 1930: Under ether narcosis the tumor was again carefully palpated and, as it seemed to be more prominent under the posterior border of the sternomastoid the skin incision was planned accordingly. When the posterior fibers of the muscle had been freed by blunt dissection and the body of the muscle retracted mesially, the tumor was found to be within the carotid sheath. This was opened and, the true nature of the growth now being manifest, slow, careful blunt

* From the Department of Surgery, University of Illinois School of Medicine. Presented before the Chicago Surgical Society, December, 1930.

dissection was carried out at the point of bifurcation of the carotid artery. Remembering Chassaignac's tubercle, we felt confident that we could rapidly compress the carotid against it should the necessity arise. It was noted that the external carotid was indented as a result of the constant pressure of the tumor. The nutrient artery with the ligament of Mayer was ligated. The adventitia of the carotids remained intact. The incision was closed without drainage; the operation lasted fifty minutes.

Postoperatively, the patient had a rise in temperature to 100°F. on the second day and complained of slight headache and pain in the neck. No mydriasis developed at any time. The skin stitches were removed on the eighth day and the patient returned home on the tenth postoperative day.

Pathological Report (Prof. Jaffé): This specimen consists of a piece of tissue 3 × 3.5 × 1.5 cm. It is soft connective tissue in which are imbedded four firmer nodules, three of which seem to be loosely connected to one another. On section these firmer areas are a yellow-grey color, very poorly defined and without definite boundaries.

Microscopical: The tumor is composed of alveoli of delicate polyhedral cells and single very large cells with hyperchromatic nuclei.

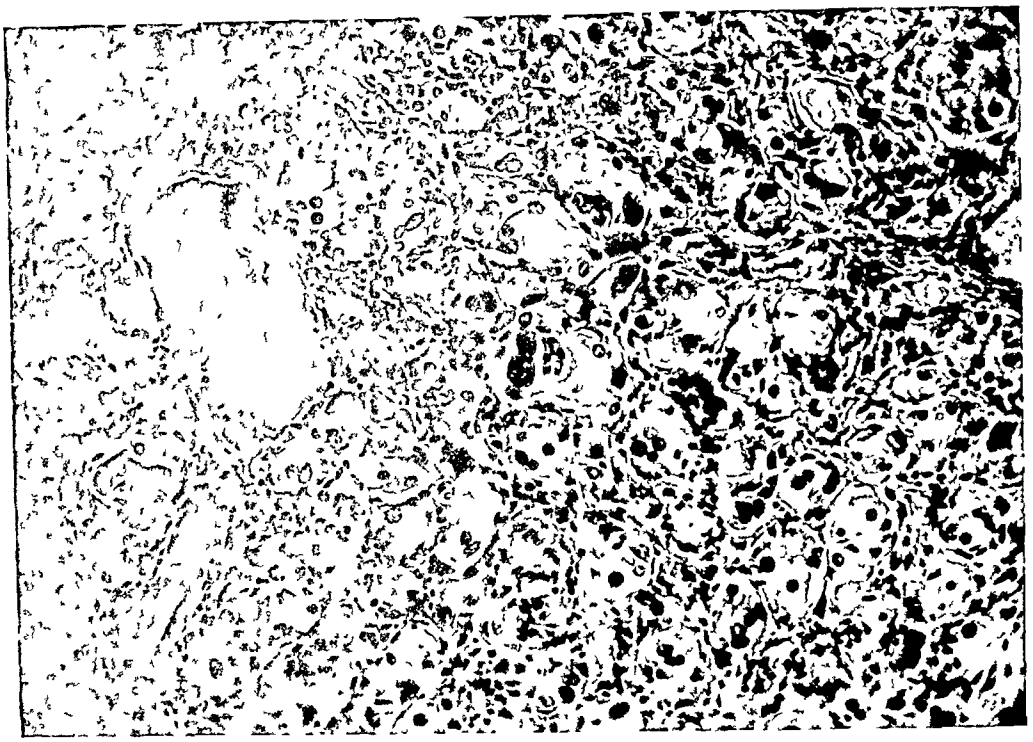


FIG. 1. Photomicrograph of carotid gland tumor.

Broad septa of a dense connective tissue with numerous mast-cells subdivide the tissue into lobules. In this stroma bundles of nerve fibers are found.

Diagnosis: Carotid gland tumor.

Our patient has reported for observation on several occasions and sixteen months after the removal of his tumor there is no sign of a recurrence and he is free from all symptoms.

That carotid body tumors are being recognized more frequently than in the past is evidenced by the fact that Wellbrock was able to collect 196 cases up to April, 1931, 53 new ones since the Bevan-McCarthy report of December, 1929. Statistics notoriously lend themselves to varied interpretations but, of the 36 cases reported in the literature during the period 1920–1928, we find that in 13 cases (36 per cent) the surgeon was able to enucleate the tumor without blood-vessel ligations. *Per contra*, among the 97 cases reported during

the period 1880–1920 only 18 (18 per cent) were removed by blunt dissection alone. Pursuing our statistics still further, we discover that approximately 50 per cent of the reported cases of carotid body tumor operations in 1930–1931 were enucleations. One may fairly safely conclude that, with improved technique and gentle perseverance 50 per cent of these tumors can be safely removed without damage to the carotid vessels, internal jugular or vagus nerve. Ligation of the external carotid alone does not, apparently, increase the mortality rate. There are records of 13 such ligations; deducting from them 2 cases in which death could be directly traced to vagus nerve injury, there was no operative mortality in the series.

PATHOLOGY

There would seem to be little or no agreement regarding the exact morphology of carotid body tumors and much divergence of opinion concerning their malignancy. Bevan and McCarthy state that "the majority of these tumors are not malignant and the patients live for twenty or thirty years or longer with tumors of the carotid body without ever becoming malignant or without their producing symptoms of moment." Rankin and Wellbrock report 6 malignancies out of 12 cases (50 per cent). Romanis and Michiner believe that:

> The carotid body is probably derived from the column of cells which ultimately develop into the sympathetic ganglia. It gives rise to two varieties of tumor:
>
> 1. Simple, highly vascular, slow growing growths which are, microscopically, peritheliomata;
>
> 2. Very malignant growths (potato tumor of Hutchinson). Microscopically these potato tumors consist of a cellular fibrous stroma which is not very vascular and contains irregular endothelial-lined alveoli, with occasional single nucleated giant cells in them. Such a tumor is often intimately attached to all three carotid arteries, while the internal and external vessels wrap around it and indent it, so as to give it a waist, and its rate of growth varies considerably. Sooner or later it will implicate the sternomastoid, jugular vein, vagus or sympathetic. It gives rise to a large lobulated, ovoid swelling, stony hard and under the upper third of the sternomastoid. It is painless and does not involve the skin. These same authors class carotid body tumors with locally malignant tumors of connective tissue such as endotheliomata and myelomata. An especial variety, arising in connection with the tunica adventitia and showing solid columns of cuboidal cells lying around vascular lumina in a fibrous stroma, is known as a perithelioma. Such tumors are found in the parotid gland (mixed parotid tumor), submaxillary gland and carotid body. Hutchinson named the latter a "potato tumor" from the peculiar greyish translucent appearance on section.

Many pathological reports of carotid body tumors fail to mention the presence of nerve cells; a few record the occurrence of giant cells. Gask and Wilson believe that these tumors are "developed as an offshoot from the sympathetic and contain cells which, like the medullary portion of the suprarenal body, are stained brown by chromic acid. A carotid gland may become the seat of a new growth, very variable in malignancy. The malignant forms rapidly infiltrate neighboring structures and are essentially inoperable." Winslow states that the carotid body is not a gland in the usual sense of the term; considered mildly malignant in its early development it tends, later in life, to undergo carcinomatous changes. Bundles of nerve fibers are always present.

Lung suggests four embryological derivations: (1) epithelium of the pharynx; (2) blood-vessel walls; (3) nerve tissue; (4) sympathetic ganglion cells of the carotid plexus.

DIAGNOSIS

Because of its rarity, an exclusive preoperative diagnosis is seldom made. The findings leading to a correct interpretation

of the nature of the tumor can be conveniently divided into (a) essential, (b) occasional.

(a) Essential:

1. A solid tumor of long standing, situated in the anterior triangle of the neck, at or near the apex of the submaxillary triangle. In size, it varies between that of a hazlenut and a hen's egg.
2. Movable laterally, but not vertically.
3. Non-expansile pulsation.
4. Not adherent to the overlying skin except in the very malignant type.

(b) Occasional:

1. Bruit and thrill on auscultation and palpation.
2. Bulging of pharyngeal wall if the growth is very large.
3. Dilatation or constriction of pupil on side of growth, depending on degree of sympathetic irritation.
4. Tenderness on deep palpation.

TABLE I

DIFFERENTIAL DIAGNOSIS OF CONGENITAL MALFORMATIONS OF THE NECK

Tumor	Age	Location	Single or Multiple	Density	Superficial or Deep	Motility	Fluctuation
Branchiogenic cyst	Congenital	Anterior triangle of neck	Single	Soft	Superficial; may extend deeply	Movable unless infection has occurred	Present
Thyroglossal cysts	Congenital	Median line below hyoid	Single	Soft	Subcutaneous	Freely movable	Usually present.
Lumphadenitis	Youth	Anterior or posterior triangle	Multiple as a rule, often matted together	Hard at first, later softer	Both as a rule	Slight or absent	Absent early.
Carcinoma	Aged	Depends on site of primary focus	Multiple nodes	Stony-hard, except in late cases with sinus formation	Deep	None	o
Sarcoma	Youth or early middle life	Angle of jaw, sternomastoid	Multiple nodes	Softer than carcinoma	More superficial than carcinoma	Early motility	o
Fibroma	30–45	Very rare in this location	Usually single	Hard	More superficial	Freely movable	o
Lipoma	Any age	No anatomic boundaries; not uniform	Single or multiple	Doughy or "woolly" soft	Superficial	Movable in all directions	o
Hodgkin's disease	10–40	Bilateral as a rule	Multiple, in chains or discrete	Softer than malignant tumors, harder than tubercular glands	Feel like lipomata but are more deeply seated	Movable	o
Syphilis	Any age	Submental or submaxillary glands. Look for chancre	Single or multiple	Hard, painless, adherent	Both	Early lost	o
Hygroma	Congenital	Inferior maxilla to clavicle; may pass to axilla or anterior mediastinum	Multiple; there may be as many as 100 pockets	Soft	More superficial than carotid body tumors	None	May fluctuate; impulse on coughing.
Aneurism	Middle age especially	Carotids especially	Single, usually fusiform	Soft	Deep as a rule	Usually none	None
Carotid body tumor	20 to 50 in 75 per cent of cases	Under sternomastoid or at its anterior border	Single, egg-shaped discrete	Firm but elastic	Deep	Lateral motility but no vertical motility	None.

5. Attacks of syncope from vagus compression.
6. Late paresis of one vocal cord.
7. Tingling sensation in neck.

DIFFERENTIAL DIAGNOSIS

As the writer has been unable to find a more comprehensive table, he feels justified in reproducing the one he evolved in 1916 (Table 1):

TREATMENT

The ideal treatment is surgical dissection of the tumor, a slow, painstaking and very delicate operation. Its success largely depends on recognition of its nature and location within the carotid sheath as soon as the skin and cervical fascia have been incised and retracted. If the surgeon is unduly fearful of tearing one of the carotids or the jugular vein, he will wisely throw a provisional ligature around the common carotid and have an assistant ready to tie the knot should the accident occur. Better still would it be for the operator to previously identify the anterior tubercle of the transverse process of the sixth cervical vertebra (Chassaignac's tubercle), and be

TABLE 1 (*Continued*)

Gurgling	Pulsation	Bruit	Specific reaction	Fistula	Course	History	Cachexia	Aspiration
None	None	None	o	May be present; may open in mouth or externally	Slow	Congenital	o	Clear or turbid fluid.
o	o	o	o	Rarely present	Slow	Congenital	o	Clear, thin light fluid.
None	o	o	Tuberculin	Late broken down sinus	Sub-acute	Tuberculous	o	Pus in late cases.
o	o	o	o	Only in terminal stage	Rapidly progressive	Primary focus	Present	o
o	o	o	o	Only in terminal stage	Progressive	o	Late	o
o	o	o	o	o	Chronic	o	o	o
o	o	o	o	o	Chronic	o	o	o
o	o	o	Blood picture, splenomegaly	o	Progressive	Irregularly progressive	Late	o
o	o	o	Wassermann positive	o	o	Chancre, etc.	o	o
o	o	o	o	Only after trauma or aspiration	Fairly rapid; extends between fascial layers esp.	May begin as a single cyst, but spreads rapidly	o	Clear, thin fluid.
Present	Expansile, delayed temporal artery pulse on affected side	Present	Wassermann positive	o	Fairly rapid	Syphilis	o	Fatal.
o	Present but not expansile	Present	o	o	Long history of slow growth	Rather sudden change to malignancy	Only very late	o

prepared to compress the common carotid against it.

The skin incision should parallel the sternomastoid muscle, either along its anterior or posterior margin, depending on which side of the muscle the tumor appears to be protruding.

Should the tumor be found to be irremovable except by section of all of the carotid vessels, the part of wisdom is to follow Bevan and McCarthy's advice, i.e. leave it alone. Ligation of the external carotid or of the external carotid and jugular vein is quite justifiable. Stewart makes the statement that: "A tumor which is found to be irremovable except by resection of portions of the vessels should be left, unless the condition of the patient warrants the severe operative risk. Syncopal attacks definitely due to the tumor, or evidence of malignancy, as shown by diminished lateral mobility, or by biopsy, would be the chief indications for this procedure."

Radiography has not been used in a sufficiently large number of cases to enable any definite idea to be formed of its value. Bevan classes his case as a cure, but only thirteen months had elapsed at the time of his report. Stewart and Birman both report failures under radium treatment and conclude that this method of therapy should be reserved only for those malignant cases which are inoperable, as its beneficial effect is too uncertain.

CONCLUSIONS

1. The successful enucleation of a carotid body tumor without traumatizing the contents of the carotid sheath is reported.

2. It would appear certain that 50 per cent of these tumors can be removed without vessel resection.

3. Ligation and resection of all three carotids is rarely, if ever, justifiable, the operative mortality being prohibitive. Ligation and resection of the external carotid, with or without removal of a section of the internal jugular vein, does not increase the operative mortality and is not followed by dangerous circulatory disturbances.

4. Radium treatment is still *sub judice* and should be reserved for inoperable malignant cases.

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SMALL FOREIGN BODIES IN THE EXTREMITIES

AN IMPROVED METHOD FOR THEIR REMOVAL*

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EVERY surgeon who has had experience with the removal of foreign bodies from the hands or feet, or the arms or legs of patients, realizes the difficulties which in one case or another will present themselves. The object is usually small, imbedded in soft tissues, but easily movable from its original situation by manipulation, pressure or squeezing. It is commonly of a dark color so that a single drop of blood in the operative field obscures it entirely. It is often so small that it is impossible to locate, unless the incision made for removal passes squarely across it. Often it cannot be felt by the touch of the gloved finger. The smaller the object the more difficult the search may be.

The more common radio-opaque foreign bodies are sewing needles, pieces of broken lead-glass, small chips of steel or other metal, short segments of wire, and grains of emery, in the hands or fingers; sewing needles or phonograph needles in the feet; insulin or hypodermic needles in the calf or thigh or in the arm.

Foreign bodies in the muscle tissue may do little or no harm if no infection takes place; often they can be left alone if the patient's peace of mind will permit it. But foreign bodies, however small, which are located in or near the tendons or ligaments of the hands or fingers are painful and interfere with the movements of the hand. Furthermore, blood vessels, nerves, or tendon sheaths can be permanently injured if they are penetrated. The functional organization of the structures of the hand is such that the slightest damage to certain parts may have permanent results. Even the minutest particle when situated in the hand may be very annoying to the patient. Usually he insists on having it removed. Foreign bodies in the foot are apt to be painful and a hindrance in walking.

It is our practice to remove all foreign bodies from the hands and fingers, and from the feet. From other localities we remove all those which are likely to be a menace to the patient.

When a small foreign body penetrates the skin and enters the deeper structures the wound of entrance frequently is so insignificant that it is indistinguishable from neighboring scratch marks, which are numerous on the hands of industrial workers. The patient may not know the exact location of entry. The direction in which the foreign body proceeded is even more difficult to establish. Often the offending object is located some distance from the spot in which it is first supposed to be.

The first and most important requisite to speedy removal of the foreign body is a knowledge of its exact location. For this purpose we stick two ordinary needles or pins through small portions of the epidermis in such a way that the pins cross each other at right angles. The point of crossing we place where we believe the foreign body to be located. These pins penetrate only the horny layer and cause no pain and no bleeding whatever if properly inserted. They must not enter the granular stratum. The needles must be clean but need not be sterilized. Figure 1 illustrates the procedure.

The next step is to take two *x*-ray views with the pins in place. One of these views is looking down perpendicularly

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on the point of crossing of the pins. The other view is taken so that one of the pins, preferably the transverse, shows in silhouette as a single point (see Figs. 2 and 3). If both of these views are accurately and carefully taken in this way, the visualization of the position and exact location of the foreign body becomes a simple matter, and the operation is rendered successful. Taking the case here presented as an example, we see that in Figure 2 the foreign body crosses the pointed limb of the longitudinal pin about 4 mm. from the crossing of the pins. One half of it lies in the lower left quadrant, the other half in the lower right quadrant. In Figure 3 we notice that the least distance between the pin and foreign body is about 3 mm., giving us exact knowledge of the depth at which it lies. Furthermore the combination of both of these views demonstrates to us the direction of the long axis of the foreign body.

By this careful step by step procedure it becomes an easy matter to visualize exactly the spot at which to make an incision that will come directly down on the foreign body when carried to the exact depth required and directed at right angles to its long axis. It becomes, in fact, more difficult to miss the object than to find it at the first attempt.

With the pins in place and the location and position of the foreign body firmly fixed in mind we take the patient to the operating room. The field is surgically prepared (with the pins still in place) and a tourniquet is applied to the base of the finger. In case the hand is involved a Martin bandage is run from the fingers to the arm and an Esmark constrictor applied above the elbow. When the foreign

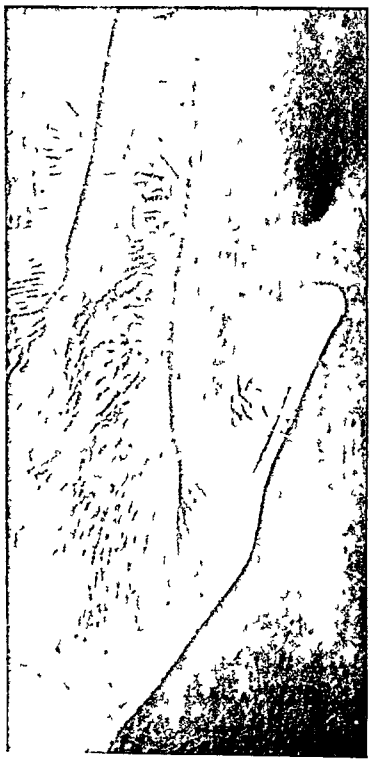


Fig. 1. Photographic view of localizing pins inserted at right angles to each other into small portion of epidermis of left thumb.

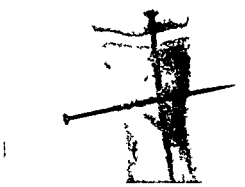


Fig. 2. Perpendicular *x*-ray view with localizing pins in place.

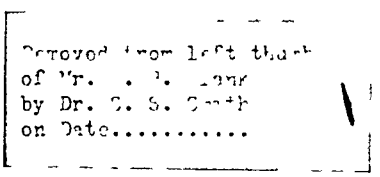


Fig. 4. Glass slide with foreign body and label affixed by adhesive tape.



Fig. 3. Silhouette *x*-ray view of foreign body in extensor tendon of left thumb, localized by crossed pins inserted into small portion of epidermis.

body is in the foot the Esmark is placed above the knee. The object is to obtain an absolutely ischemic operative field, which is exceedingly important. If there is the slightest amount of blood present the foreign body will be obscured by it. The smaller the object and the more it approaches a spherical shape, the surer the operator must be to cut down squarely upon it. A miss of a millimeter can be enough to make an otherwise easy operation not only complicated and difficult, but protracted and tedious.

The pins are removed when the surgeon is ready to make the incision and after he has the exact point located where a minute incision will lay bare the foreign body. Usually a grating sensation can be felt as the blade passes over the foreign object. Retraction, if necessary, is best obtained by opening the blades of a curved Kelly forceps in the wound. In a completely ischemic field the dark foreign body is easily seen lying in the incision, and can be lifted out of the wound without manipulation or contamination of the tissues. We prefer to use general gas anesthesia. This insures complete relaxation, which is especially important when tendons or muscles are involved. The wound is closed with a single stitch, and dressed.

The entire operation, when carefully planned and properly executed, requires only a few minutes. At no time does the operator introduce his fingers or any other object into the wound except the few instruments he actually uses. The incision usually heals promptly by primary union. We have never found it necessary to resort to the use of a fluoroscope or an electromagnet.

In Figure 4 we show a convenient and simple method of filing away small foreign bodies in an ordinary slide box. The foreign body and a properly inscribed label are placed side by side on a microscope slide and an adhesive strip applied. We keep on file indefinitely those foreign bodies which might be called for as evidence in litigation proceedings.

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* Continued from page 260.

DIAGNOSIS OF ADVANCED ABDOMINAL PREGNANCY*

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IN a former article I reported a new method of diagnosing advanced abdominal pregnancy. Since that report of the first case I have used the procedure again with much help and desire to incorporate a report of the second case. I have seen no reference in the literature to the effect that the method has yet been used by others.

I desire again to call attention to the very great difficulty which often arises in differential diagnosis between intra-uterine and extra-uterine pregnancy.

To the physician who has not had several cases of advanced abdominal pregnancy to diagnose, it may seem that the diagnosis should not be difficult. True indeed there are some cases where the diagnosis is easy, but there are other cases where the previous methods of diagnosis have been quite insufficient and the obstetric attendant is left in serious doubt as to how to proceed with such cases.

Direct palpation of the abdomen is by no means dependable. I have seen several cases where superficiality of fetal parts seemed to indicate that the fetus was not within the uterus, in which subsequent vaginal delivery proved the lack of dependability of this sign. In many abdominal pregnancy cases the abdomen is so tender and rigid that instructive palpation of the fetus is precluded.

Have furthermore been called to see three cases where the introduction of a sound into the cervix failed to ascertain definitely whether the pregnancy was within or without the uterus. Even though the pregnancy may be extra-uterine, the uterus and its cavity are often much elongated so that the sound seems to discover a uterus much beyond the average non-pregnant size.

In each of these three cases a bag had been introduced into the cervix in an effort to obtain dilatation only to find that the fetus was not within the uterus.

The *x*-ray has failed me now in 8 cases. To be sure the *x*-ray shows the fetus, but it fails to demonstrate whether the fetus is inside or outside the uterus. It must be remembered that in abdominal pregnancy there is developed a false sac and some water, and the roentgenogram often shows this sac and water surrounding the fetus, but it looks too much like the normal pregnant uterus for a definite decision.

In my first case reported, the *x*-ray showed a fetus of approximately six months and a fetal heart beat was obtainable, yet the roentgenologist was quite unwilling to go on record as to whether the fetus was within or without the uterus. As the patient was in a serious condition it was quite necessary to arrive at a diagnosis. Iodized oil was introduced into the uterine cavity and the *x*-ray picture plainly showed the usual triangular uterine cavity as seen in non-pregnant uteri, thereby absolutely proving the fetus must be outside the uterus. Laparotomy was resorted to at once and corroborated the presence of an advanced abdominal pregnancy. (Details are published elsewhere.[1])

Since the report of this case another has come to my wards which presented the usual difficulties in arriving definitely at a diagnosis, there being much difference of opinion among consultants. An *x*-ray picture again showed a fetus of about seven months, probably macerated, but failed to determine satisfactorily whether the fetus was within the uterus or within the false sac of an advanced abdominal pregnancy. In view of the serious condition of the

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patient a definite diagnosis was very important. Iodized oil was introduced into the uterine cavity and an *x*-ray



Fig. 1.



Fig 2.



Fig. 3.

Fig. 1. Lateral *x*-ray showing fetal skeleton but giving no definite information as to whether fetus is intra-uterine or extra-uterine.

Fig. 2. Same patient as Figure 1; anteroposterior *x*-ray showing fetus but failing to demonstrate conclusively whether fetus is intra-uterine or extra-uterine.

Fig 3. Same patient as Figures 1 and 2 after injection of iodized oil into uterus, showing usual triangular uterine cavity of non-pregnant uterus. Fetus can also be seen.

picture taken which quite conclusively showed a non-pregnant uterine cavity, leaving the only possible conclusion of advanced abdominal pregnancy. Laparotomy was done promptly and the diagnosis further confirmed.

The introduction of iodized oil into the uterine cavity followed by an *x*-ray picture is by no means new. It is being done in thousands of cases for many conditions such as sterility, fibroid tumors, etc., and in a relatively few instances this procedure has been resorted to in an effort to diagnose normal uterine pregnancy,[2] but a search of the literature fails to reveal its use for differential diagnosis between uterine and extra-uterine pregnancy.

Such a procedure for the diagnosis of normal uterine pregnancy does not seem justifiable in view of the great danger of abortion,[3,4,1] and with the present development of the Ascheim-Zondek test for pregnancy it would seem quite unnecessary to resort to such an examination when the diagnosis can be so certainly established without risk.

But for the cases, of which there are many, which present serious difficulty in definitely determining whether the pregnancy is within or without the uterine cavity, the procedure herein reported offers a very dependable diagnostic means.

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END-TO-END APPROXIMATION & ACCURATE REDUCTION

AS A NECESSITY IN FRACTURE THERAPY*

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IN this day of the roentgenogram, that positive piece of evidence by which the surgeon is often misjudged,

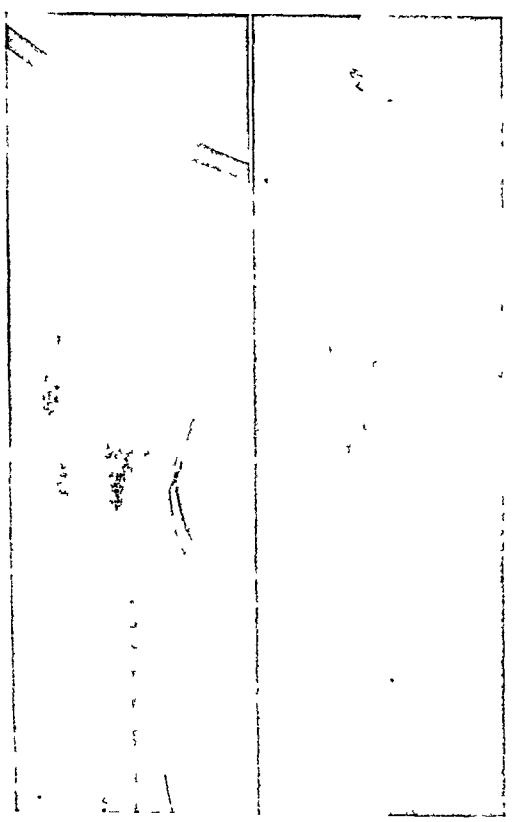


FIG. 1A. FIG. 1B.

FIG. 1. L. S., female, aged eighteen. Comminuted fracture of mid-shaft right femur. Automobile accident. View (a) day of admission Sept. 5, 1929. (b) Nov 28, 1930, approximately fifteen months after injury. Traction therapy. No shortening and perfect functional result. Playing tennis.

one is frequently confronted with the question of end-to-end approximation and necessity of accurate reduction in the treatment of fractures of bones. Many times conclusions are indefinite. Oft-times, despite careful and timely attempts at reduction, one is forced to accept an approximation that is anything but an accurate reposition of the parts involved. Immediately the question arises as to the ultimate result. Will there be firm union? Will there be deformity? What can one expect as to return of function? Will the patient be able to resume his previous occupation? These and many other questions the surgeon asks himself. He is interrogated to no end by the patient. It is needless to say that if all fractures were readily reducible, were easily maintained, and would unite firmly with an ultimate good result, satisfactory to patient and physician alike, this discussion would have no place.

AIMS IN FRACTURE THERAPY

The major aims in all fracture therapy are fundamentally twofold. One first attempts to obtain accurate reduction.[1] Accurate reduction as defined by the fracture committee of 1915 signifies anatomical restitution of fragments to the position they occupied before the fracture occurred, and their maintenance in that position until consolidation has taken place. Is it always possible to obtain accurate reduction? Can this aim be satisfied to its fullest meaning in all fractures? The reply is quite obvious.

The second consideration is the restoration of function. Of what use is a limb whose function has been lost because of injudicious attempts at reduction? What justifies added risk to life in the repeated induction of anesthesia? What justification is there for repeated mauling of a limb in attempts at accurate reduction, when by the anesthesia or these manipulations a life may be actually lost? It is a

* From the surgical service of Dr. E. L. Eliason at the Hospital of the University of Pennsylvania.
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known fact that repeated attempts at reduction oft-times result in non-union. As recently stated by Ashhurst:[2] "Mal-union of a moderate degree is less evil than non-union. It is better to leave moderate mal-union than to secure reduction at the expense of firm union."



Fig. 2A. Fig. 2B.

Fig. 2. O., male, aged eighty-five. Fracture of surgical neck of right humerus. Cardiac decompensation and asthma precluded rational therapy. Treated in ambulatory manner with no reduction other than maintenance of axis. Back at work with comfort in six weeks. A. Immediately after fracture. B. Ultimate result.

METHODS FOR ATTAINING ACCURATE REDUCTION

The surgeon has at his disposal several possible courses of action to attain an accurate reduction. Briefly stated, these methods are: (1) Closed reduction, with or without fluoroscopic control, manipulation and locking of fragments, which are maintained thereafter by proper splints. (2) Open reduction with or without internal fixation, and in either instance maintenance of position by proper splints. (3) Continuous traction, commonly used in fractures of the femur, humerus and tibia. The assumption of any particular course depends primarily upon the individual fracture. The election of a method frequently is determined by one's limitations, lack of equipment and inability to perform open operation. When closed methods of reduction fail to bring position to accuracy, is one justified in recommending open operation, with its risks, both immediate and remote, to attain accurate approximation? Barring ordinary contra-indications to operation, when are we permitted to recommend open reduction?

SATISFACTION OF THE PATIENT

The general concept of fracture therapy has been altered to some extent by a public only too well aware of the possibilities of a malpractice suit. Often, despite personal negligence, the patient attempts to throw the entire blame of an injury with its incurred expense, coupled with the cost of maintaining his dependents for that period of disability, upon some second party. It matters little to him whether he is recompensed by an insurance company, a workman's compensation fund, or a physician through malpractice suit.

One must always strive to satisfy the patient. If the patient is dissatisfied by a deformity, mal-union or even slight loss of function, even though he has been warned at the onset of the treatment, he may turn on us with all the weapons of a recently acquired medical or legal advisor. The principal factors on which judgment may be based are the *x*-ray, with the roentgenogram as its permanent

record; the actual deformity and loss of function.

The most prominent weapon is the *x*-ray, which we have used to advantage in the actual diagnosis and treatment of the fracture, and which is now turned against us. The *x*-ray is responsible for more attempted open reductions than any other single factor. By the roentgenogram the position of the bony fragments can be accurately determined, and after reduction, by this means are we judged.

Very few reductions can be considered satisfactory, as one often discovers to his discomfort, unless controlled by *x*-ray examination, repeated as each case indicates. In the absence of the roentgenograms, actual deformity and loss of function are formidable weapons in the hands of a dissatisfied patient and unscrupulous lawyer. When the roentgenograms are available, deformity and loss of function may be interpreted in their light.



FIG. 3. H., male, adult. Fracture of upper third of radius. Illustrating failure to restore axis to normal, and to properly maintain reduction. Bad result.



FIG. 4. L. B., male, aged forty-seven. Simple fracture of upper third of shaft of femur. Buck's weight extension for six weeks. *X*-ray proved end-to-end apposition at that time. All extension then removed, and shortly thereafter patient complained of pain at site of fracture. Discharged from hospital without further x-ray examination.

Present view taken six months after accident, at which time patient applied to this clinic for operative reduction because of bowing and shortening of about 1¾ inches. Firm bony union made operative intervention inadvisable.

SUFFICIENT REDUCTION

What degree of reduction is sufficient? The answer is multifold, and depends upon (1) the site of fracture, (2) the age of the patient, and (3) the function required. In the long bones, namely the humerus, the femur, and the upper two-thirds of the tibia, and more rarely both bones of the forearm, the following criteria may be stated: (a) restore the axis of the fragments to normal, (b) prevent appreciable shortening, and (c) obtain bony

union. There are exceptions to these criteria which will be considered.

Figures 1 and 2 are illustrative of good remodeling, and according to Ashhurst[2] initial shortening of 1 cm. will be corrected, above which appreciable shortening will



Fig 5A. Fig 5B. Fig 5C.

Fig. 5. Male, aged thirty-six. Picked up by police in rum runner accident. Incarcerated. Prison physician applied a single forearm splint, not including elbow or wrist. Patient brought to hospital thirteen days later with result shown in A and B. Open reduction accomplished result shown in C. Patient was discharged with union five weeks later.

results where the foregoing criteria have been satisfied.

Figures 3 and 4 are illustrative of poor results where the same criteria have not been satisfied.

EXCEPTIONS

In the young, there is, especially in the long bone fractures, extensive remodeling and subsequent overgrowth of bone due to stimulation from the injury which often will compensate for shortening. Displacement is corrected under Wolff's law, in that "form depends upon function." The margins are more limited in the adult, as there is not such rapid and extensive occur. Children more readily compensate for uncorrected deformities.

NECESSITY OF ACCURATE REDUCTION

Accurate reduction becomes more essential when the fracture line is near or involves a joint. Accurate reduction, at least end-to-end approximation, is most essential when one bone of the forearm is involved, becoming a necessity during the growth period. The areas in which accurate reduction must be carefully considered are: (a) At or near the shoulder joint. Exceptions enter here, because the movement of the scapula may compensate for loss of function of the shoulder joint.

However, no reasonable effort should be spared to secure accurate approximation. (b) Elbow joint, about the head and neck to restore the line of the wrist joint, which when disregarded results in a weak and oft-times useless wrist. (h) Both



FIG. 6A. FIG. 6B.

FIG. 6. Female, young adult. Supracondylar fracture of humerus (A) prior to closed reduction; (B) after reduction and application of suitable splint. Good result. .

of the radius and displacement of the condyles of the humerus. (c) About the lower end of the radius, where deformity may interfere with function. (d) Intracapsular fracture of the neck of the femur, where non-union is apt to occur. (e) About the ankle joint, or lower one-third of the tibia, disregard of which may result in lasting disability from foot strain. In these fractures special attention should be focussed to the preservation of the mortise of the ankle joint and alignment of the astragalus with the tibia. (f) About the knee joint, although exceptions have been noted. (g) Where one bone of the forearm is involved, reduction is necessary bones of the forearm, so that function may be restored to a maximum.

Figures 5 and 6 are illustrative of good results where the foregoing factors have been observed.

RESPONSIBILITY AND THE PATIENT

It is again evident in the last group that all attempts at accurate reduction are aimed at preservation of function. The surgeon cannot be criticized when, failing to attain reduction of a fracture by the closed method, he advises open operation, knowing that he possesses the skill to improve the position of the bony fragments, and is then refused. The

responsibility there rests with the reliable patient or parent. When comminution of fragments complicates a fracture, open reduction is often questionable.

WRITTEN RECORD

In order that there may be no misunderstanding between the patient and surgeon a written record is most advisable. It has been our policy during the past few years to state exact aims to the patient. These objectives with their possible limitations are produced in writing, supported by reliable witnesses, and attached to the record. When possible the patient's signature is added. When, as in a fracture of a long bone, traction will satisfy our three aims (axis, length and union), the patient is so informed. If, when an open operation is advised, the objectives and the limitations are clearly stated, there can be no misunderstanding. Should a patient refuse an essential operation, he should be made cognizant of the probable ultimate outcome. These statements should be attached in writing with the patient's signature affixed. The responsibility at this point is most certainly his. When in doubt as to the necessity of accurate reduction or end-to-end approximation, it is much wiser to share the responsibility with one more experienced in the treatment of fractures.

POSSIBLE LIMITATIONS TO THERAPY

Aside from the fact that a fracture may in itself, because of its compound character, comminution or soft tissue injury, offer definite limitations to therapy, there are extraneous factors sufficiently important to merit consideration. These may be classed:[3] the patient, the surgeon and the operating room.

The Patient. Shock is the only contraindication to immediate reduction. No patient suffering from hemorrhage, cardiac, renal, hepatic or pulmonary disease or senility should be lightly subjected to actual operative measures. Difficulties may be surmounted by strict and careful preoperative preparation. The thoughtful selection of an anesthetic is quite essential. Alcoholism and drug addiction may be contraindications to operation. Pathologic fractures or spontaneous fractures should be carefully scrutinized to determine their possible etiology before subjecting them to actual operative measures.

The Surgeon. Experience is most essential in the surgeon. He should likewise possess ability and ingenuity. In this work as in any specialized field, an apprenticeship is most desirable. Extensive training can only be obtained opposite an experienced surgeon.

The Operating Room. Fracture work should only be undertaken when the operating room, including the force of assistants, nurses and orderlies are especially fitted for the work. Rigid asepsis is most essential. The Lane technique is desirable. The supply of instruments and apparatus must be complete and in working order. Operative work on fractures, especially where much traction and manipulation is needed, is extremely shocking to the patient, and time saved means lessened shock.

INDIVIDUALIZATION

Despite attempts to classify or standardize the treatment of fractures, each fracture is a law unto itself, and should be treated individually. The reduction sufficient for one who never uses his extremities cannot be compared to that demanded for one whose livelihood depends thereon. The risk to the patient must be justified by the objective. Individuality of treatment extends from the time of actual reduction with fixation to the resumption of full function. Individuality is required of the surgeon. The same individual should be responsible for the treatment of the fracture from the time of reduction to the resumption of function. The person caring for any fracture should personally review all roentgenograms made of that particular

fracture, before, immediately after reduction and as often thereafter as each case justifies.

To the patient, we cannot be condemned for giving justifiable advice; more often are we criticized for failure to give any.

SUMMARY AND CONCLUSIONS

1. That degree of reduction termed sufficient depends upon: (a) site of fracture; (b) age of the patient; (c)) required function.

2. In the long bones, namely the humerus, the femur, the upper two-thirds of the tibia and more rarely both bones of the forearm, the following criteria may be stated: (a) restore axis to normal; (b) no appreciable shortening; (c) obtain bony union.

3. Accurate reduction becomes more essential in the following areas: (a) near the shoulder joint; (b) at the elbow joint, head and neck of the radius; (c) lower end of the radius; (d) intracapsular fracture at the neck of the femur; (e) about the ankle joint; (f) about the knee; (g) one bone of the forearm; (h) both bones of the forearm.

4. Avoid legal complications by: (a) preoperative roentgenogram; (b) postoperative roentgenogram; (c) written record to contain the following essential points: (1) permission to treat; (2) actual statement of aims when operation is advised, with a summary of the possible limitations. Affix the patient's signature to his acceptance or rejection of such advice.

5. Definite limitations to therapy may be classed under the actual headings of the patient, the surgeon, and the operating room.

6. Fractures should be individualized from the following angles: (a) the patient, his occupation, (b) procedure of reduction; (c) fixation; (d) after-treatment; (e) the surgeon.

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WATER & CHEMICAL BALANCE IN SURGERY*

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SINCE water and sodium chloride are both essential to life, it is quite self evident that any deficiency in these elements beyond the minimal requirements will cause disturbances in the chemistry of the body, resulting in disease. In certain disease conditions, there is a loss of water and salt by the body, and a readjustment of these essentials will result in improvement or cure. It is important that careful attention be given to deviations from normal in the chemistry of the body as a part of all rational surgical treatment.

There is a definite circulation in and out of the upper intestinal tract that has great importance in the study and treatment of disease involving this region. Rowntree[1] has tabulated the quantity of secretions entering the intestinal tract in twenty-four hours, as saliva, gastric juice, bile, pancreatic juice and succus entericus and estimates that it amounts to 7500 and 10,000 c.c. This is two to three times the quantity ordinarily ingested by mouth, three to four times the quantity secreted as urine and about twice the total volume of the blood. It can readily be seen that any obstructing lesion of the upper intestinal tract, which prevents the reabsorption of the water and chemical elements secreted, may result in serious loss of essential constituents of the body in large quantity.

Of the disturbances incident to preoperative and postoperative treatment, the surgeon is chiefly concerned with dehydration, hypochloremia and acid-base balance. While it would be difficult indeed to tabulate the many roles played by water in the intricate chemistry of the body, it may serve for clinical purposes to point out some of the well-known functions which depend upon water balance. It has been shown experimentally that by starvation an animal can lose glycogen, fat and protein to 40 per cent of its body weight, but a loss of 10 per cent of the water content of the body results in serious disorders and a loss of 20 to 22 per cent results in death. The proper quantity is necessary for the normal functioning of every living cell. Generally this quantity is fairly constant. It plays an important role in heat regulation, physicochemical properties, such as solvent power, hydration, ionization, imbibition and surface tension, lubrication of serous surfaces, elimination and is essential for proper growth of the organism.[2] Sodium chloride is essential in maintaining acid-base balance, osmosis and water distribution, formation of hydrochloric acid and the gastric juice and may be a factor in maintaining the muscular tone of the intestinal tract[3] and in the proper growth of the organism.[4] According to Fleming[5] the bactericidal power of the blood is influenced by its sodium chloride content.

The clinical terms "alkalosis" and "acidosis" are often rather loosely used and frequently poorly understood. For practical purposes an estimation of the carbon-dioxide combining power of the blood will give the evidence necessary to indicate acid and alkaline changes which are important in the recovery from disease. To wait for an expert chemist to estimate changes in the hydrogen-ion concentration of the blood would be to postpone treatment until the patient reached extremis.

In a study of the water and chemical balance in preoperative and postoperative surgical conditions, dehydration, hypochloremia and acid-base balance must be considered together, since they are intimately associated in the pathological processes present and are treated together

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Table I

Alkalosis may be Suspected When There is (Cutting):

1. Dehydration
2. Blood concentration shown by increase of red cells and hemoglobin
3. Nausea and vomiting
4. Low blood pressure
5. Marked asthenia
6. Increase of blood non-protein nitrogen, urea and creatinine
7. Albumin and casts in urine.

Acidosis may be Suspected When There is:

1. Drowsiness and sluggishness
2. Irritability
3. Hyperpnea
4. Nausea and vomiting
5. Headache
6. Abdominal pain
7. Dehydration
8. Low urinary output
9. Cyanotic or cherry red lips
10. Convulsions
11. Coma.

as a group or symptom complex. Experimental dehydration[6] has shown a terminal rise in the non-protein and urea nitrogen, an elevation of the blood chlorides, an increase in the fibrinogen content of the plasma and an increase in the total protein of the blood serum. In acute high intestinal obstruction there develop pronounced chemical changes involving dehydration, hypochloremia and acid-base balance. There is an abnormal destruction of body protein as shown by the increase in non-protein and urea nitrogen in the blood. There is a change in the blood toward the alkaline side, as shown by an increase in the carbon-dioxide combining power. There

Table II

Conditions Showing Decrease in Blood Chlorides

1. Obstruction of esophagus and cardia (experimental)
2. Obstruction of pylorus
3. Upper intestinal obstruction
4. Gastric, duodenal and upper jejunal fistulae
5. General peritonitis
6. Pneumonia
7. X-ray
8. Severe burns
9. Anaphylactic
10. Gas poisoning (early stages)
11. Miner's cramp.

is a fall in the blood chlorides due to a loss of this element from the body. It is interesting to note that in obstructive lesions of the stomach and upper intestinal tract there develops an alkalosis as a result of vomiting and in infants an acidosis may develop, as a result of severe diarrhea. This is explained by the loss of hydrochloric acid in the first instance and loss of alkalies from the upper intestine in the second. Elman and Hartmann[7] have shown that a complete loss of the pancreatic juice will result in acidosis unless there is associated some vomiting to increase alkalinity by loss of acid.

Complete loss of the gastric juice[8] or complete loss of the pancreatic juice will result in death in a few days with changes in the blood chemistry similar to those found in high intestinal obstruction. Death will also result in two to five days with experimental complete drainage of the upper jejunum.[9] The similarity of the blood chemical changes in these conditions to those in intestinal obstruction leads to the conclusion that the cause of death in the latter condition may be due to a loss of the upper intestinal tract secretions by vomiting or accumulation in the stomach and upper gut.

Underhill and his associates[10] have made some interesting observations on the changes occurring in the blood in superficial burns. They have found that the blood becomes highly concentrated with a hemoglobin percentage that may be twice the normal. In spite of this extreme concentration of the blood, the chlorides show a decrease as noted by Davidson.[11] The importance of reestablishing the water and sodium chloride balance in extensive burns is obvious.

The low chlorides found in the blood and urine of pneumonia patients has suggested a replacement treatment with sodium chloride. Haden[12] and Sabatini[13] have each reported beneficial results by such treatment.

The rather startling observation by Rowntree[1] that animals can be intoxicated by giving them excessive doses of water and that this intoxication can be prevented or cured by administering hypertonic sodium chloride solution is an excellent

lesson in water and salt balance. Brockbank[14] describes a condition existing in miners, who are exposed to considerable heat, which he calls "miner's cramp." He attributes the cramp-like pains which are present in the arms, legs and abdomen to loss of water and sodium chloride by excessive perspiration. When such patients drink salt solution the cramps are cured or prevented.

In the treatment of any disease in which there exists a water or chemical imbalance it is obviously desirable to have some means of estimating the quantitative needs. The quantity of water loss is difficult or impossible to estimate accurately. The guides of most value are thirst, a knowledge of the daily intake, daily output in the urine, a knowledge of the quantity that it is possible to lose from the upper intestinal tract, and a determination of blood concentration by hemoglobin or hematocrit readings. If thirst exists and the patient has failed to ingest and excrete the normal quantities, water is, of course, needed to return these conditions to normal. Underhill[10] states that an animal or man cannot long survive blood concentrated to 140 per cent of the normal value. Quantitative estimations of the blood chlorides and acid-base balance can now be made with accuracy in the laboratory. In addition, study of the non-protein and urea nitrogen and the creatinine of the blood gives very valuable information concerning the condition of the patient and the prognosis. As long as abnormal readings exist in the blood chemistry, treatment is indicated. Such readings are, therefore, of great value as a therapeutic guide.

In the treatment of patients having obvious water and salt deficiency, it is not sufficient to instruct the assistant, intern or nurse to "force liquids," or to give saline solution. An accurate liquid chart, showing the daily intake and output, should be kept and in the very ill, blood chemical readings daily should be compared. The treatment instructions should, therefore, be based upon definite findings at the time and continue to be based upon the daily needs of the patient as estimated clinically and in the laboratory.

Any acid-base disturbance is so intimately associated with water and salt imbalance that the treatment of the latter usually suffices to correct the former. The alkalosis which develops in pyloric and high intestinal obstruction is relieved by giving sodium chloride solution. In acidosis glucose is added to the water instead of, or in addition to, sodium chloride. The administration of a solution of sodium bicarbonate to relieve acidosis and acid sodium phosphate to relieve alkalosis is rarely necessary and not without danger unless very carefully controlled.

The quantity of water, salt or glucose necessary to restore balance, obviously depends upon the need of the patient at a given time. In extreme dehydration, as a result of long water deprivation or persistent vomiting over a long period of time, the initial supply of water should exceed several times the average daily intake. Extreme sodium chloride loss should be promptly corrected by administering a hypertonic solution of this salt in sufficient quantity to restore the blood chlorides to within normal limits. Following this initial treatment within the first twenty-four hours, the normal balance may usually be maintained by administering physiologic sodium chloride solution, glucose solution and tap water.

Distilled water and tap water should not be used under the skin or in the vein. A 1 or 2 per cent sodium chloride solution or a 3 per cent glucose solution may be given satisfactorily by hypodermoclysis. Concentrated solutions given rapidly under the skin may cause sloughing. The administration of distilled water alone is of little clinical value when there exists a marked disturbance of sodium chloride or acid-base balance.

To maintain water balance in the average postoperative patient for the first few

days, a minimum daily intake of 3 liters of liquid may be satisfactorily used as a working basis. This is slightly in excess of the normal average daily adult intake. It may be necessary to increase or decrease this quantity, depending upon the clinical condition and progress of the patient.

In acute conditions with vomiting and low blood chlorides, salt therapy may be started with 500 c.c. of a 5 per cent solution given in the vein. This quantity, supplemented by physiologic salt solution under the skin, in the vein and by rectum, given in sufficient quantity to supply the water needed, will usually suffice to maintain sodium chloride at a normal level in the blood. Excessive quantities of sodium chloride solution may produce general edema. For the treatment of acidosis and to furnish food, glucose may be satisfactorily given intravenously in 10 per cent solution alone, or in combination with salt solution. In no sense is glucose a substitute for sodium chloride in the treatment of disease in which there exists a hypochloremia.

Generally speaking, it is wiser to give hypertonic intravenous solutions in doses not exceeding 500 c.c. However, continuous "venoclysis" may be given, providing the rate of inflow is very slow. The work of Hirshfeld, Hyman and Wanger[15] has emphasized the importance of giving intravenous solutions very slowly to avoid what they have termed "speed shock." They believe that reactions rarely occur when intravenous therapy is given at a very slow rate. In accord with this viewpoint they recommend that a solution of more than 100 c.c. in quantity be given at the rate of 2 to 3 c.c. per minute and less than 100 c.c. at the rate of 1 c.c. per minute. For practical purposes we have found the following rates of intravenous administration quite satisfactory:

1000 c.c. of physiologic sodium chloride solution in 1 hour
500 c.c. of hypertonic sodium chloride solution in 1–1½ hours
20 c.c. of 10 per cent sodium chloride solution in 5 minutes
500 c.c. of a 10 per cent glucose solution in 1–1½ hours
50 c.c. of a 50 per cent glucose solution in 10–15 minutes

Continuous intravenous drip 30–60 drops per minute.

SUMMARY

In order to obtain the best surgical results each individual patient should be studied from the standpoint of water and chemical balance. Certain diseases, such as high intestinal obstruction and severe burns, which show marked water loss and chemical changes, are dependent for cure upon a careful estimate and correction of the water loss and chemical imbalance. To restore to the body its normal constituents, which have been lost by disease, is a most logical type of therapy.

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CARCINOMA OF THE PENIS*

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THIS case of carcinoma of the penis is of interest because of its apparent association with a circumcision.

Mr. F. G., a Porto Rican, forty-three years old, married, was first seen at the Urological clinic of the Long Island College Hospital on August 1, 1930 complaining of soreness of the penis. He gave the following history: He was circumcised ten months ago. Since that time the glans penis had been persistently red and tender, and would not heal. He had been unable to indulge in sexual intercourse since then because of the associated pain.

Examination revealed the glans penis to be entirely covered by a somewhat irregular, red, moist tissue resembling granulation tissue. This extended from the meatus to the region of the corona at which point the skin from the shaft of the penis was adherent. The glans was also found to be quite tender.

It appeared as though the mucous membrane of the prepuce had been adherent to the glans and that at the time of the circumcision only the skin had been removed.

He was advised to undergo an operation which intended to free the adherent granulation tissue and expose the normal surface of the glans. Six days later this was attempted under general anesthesia. A meatotomy was first performed because of a very narrow meatus. A line of cleavage was then sought for at the meatus, but this was almost impossible to obtain. By both sharp and blunt dissection this superficial tissue was separated from the glans and stripped back to the corona, exposing for the most part a smooth glans penis. The skin was then incised at the corona, freed for a distance of one inch, and sutured to Buck's fascia just proximal to the corona. This left the glans with a free corona. When the patient was discharged six days later, the surface of the head of the penis had begun to show the formation of epithelium. Two months later he was able to indulge in intercourse without pain or discomfort.

He was kept under observation in the clinic and on September 18, just six weeks after the operation, a warty growth on the left



FIG. 1. FIG. 2.

FIG. 1. Epithelioma of penis. Lesion is at tip of glans.
FIG. 2. Nine months following partial amputation of penis.

and dorsal region of the tip of the glans was noticed. It gradually increased in size and hardness until in January, five months following the plastic operation, it covered an area 2.5 by 1 cm. This was markedly indurated and cornified, and was elevated about 3 or 4 mm. above the surrounding surface. On three separate occasions this growth was cauterized, but with little or no improvement. Later a biopsy specimen removed by the radiotherm knife was reported by the pathology department to be an epithelioma.

Because of the localized involvement and the early stage of the growth, it was thought that a conservative amputation at about the midportion of the shaft of the penis would suffice to give as good a prognosis as a more radical procedure. And as there was no evidence of involvement of the inguinal lymph nodes

* From the Department of Urology, Long Island College Hospital, Brooklyn, N Y.
Read before the Brooklyn Urological Society, December 8, 1931.

surgery was preferred to either *x*-ray or radium.

On March 27, seven months following the previous plastic procedure, a partial amputation was performed under spinal anesthesia. This was done in the usual manner using the radiotherm knife to amputate the corpus spongiosum, and also the corpora cavernosa after the latter were ligated individually with chromic catgut. The skin was sutured in a vertical direction, and the urethra which was left somewhat longer than the corpora cavernosa was sutured to the lower angle of the skin wound. A winged tip catheter was placed into the bladder to divert the urine. This was removed after five days and the patient was then taught to pass a short glass sound daily to avoid stricture formation at the meatus. He was discharged from the hospital fourteen days following the operation with the wound healed except at the lower angle adjacent to the meatus.

Two weeks following his discharge from the hospital it was noted that the lower angle of the wound still remained unhealed. Because of the suspicious appearance of the tissue, a biopsy was performed. This was reported to be only infected granulation tissue.

Six weeks following the operation, deep *x*-ray therapy was begun as a prophylactic measure. This consisted of three courses of treatment totalling twelve exposures to both inguinal regions. This was completed last September.

It is now nine months following his operation. The stump of his penis although it may appear small in the photograph is able to carry on all its functions; he voids a good stream and is able to direct it, and he has satisfactory intercourse. The skin is still adherent to the distal end of the corpora which causes an upward bowing on erection; otherwise he has no complaints.

COMMENT: Apparently this patient at one time had an adherent prepuce and at the time of the circumcision the adhesions between the surface of the glans penis and the mucous membrane of the prepuce were not broken up, and only skin was removed. The new surface of the glans then became chronically infected and constantly irritated.

This may or may not have had a relationship to the carcinoma, but if irritation may be considered one of the etiological factors in cancer, we have a competent cause here.

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REPAIR OF CRANIAL DEFECTS*

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INDIANAPOLIS, IND.

WITH our increased knowledge of the treatment of head injuries we are saving the lives of an increasing number of this type of patient. Many of these individuals, however, after recovering from their acute condition are doomed to a life of chronic invalidism or suffer various degrees of physical and mental impairment. To prevent or to combat these disabling sequellae following head injuries is one of our most important problems. One type of sequella that may be very disabling is the loss of a part of the cranium following a compound fracture of the skull. Many persons are struggling under this handicap of a cranial defect who could be easily relieved by a cranioplastic operation. This operation is so safe, so easy to perform, and in most cases gives such satisfactory results that every adult patient with such a defect should be given the benefit of a plastic repair. The two most generally accepted operations are here reviewed in the hope of stimulating a more frequent use of these procedures to rehabilitate this class of patients. While these operations are often tedious and time-consuming, they can be performed by the general surgeon. Moreover, they do not require special instruments or equipment. The results following the repair of a cranial defect are most gratifying, and I have yet to see a patient who, if not entirely relieved of his symptoms, was not at least benefited to a marked degree.



FIG. 1. Patient on admission to Robert Long Hospital.

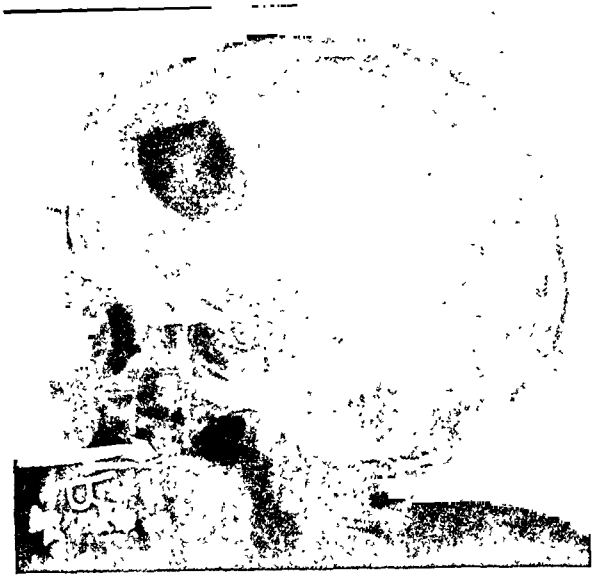


FIG. 2. *X*-ray of skull showing bone defect.

The most prominent symptoms complained of by patients with a cranial defect are headache and dizziness which becomes worse on physical exertion and with changes in barometric pressure. Some are greatly annoyed by the constant sensation of pulsation at the site of the defect. If the defect is over a motor area there may be attacks of Jacksonian epilepsy. Besides

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these physical symptoms there is generally a justifiable degree of neurosis due to the loss of part of the bony protection of the



Fig. 3. Patient on dismissal.

brain. The patient is in constant fear of being struck over his soft spot, and he fears leaving the shelter of his home. In the most stolid individual, feeling the pulsations of one's brain just beneath the scalp would be at least disquieting. The neurotic symptoms may be many and varied. In some patients the neurosis may show itself in the form of local or generalized convulsive attacks but it is seldom difficult to differentiate the neurotic from the Jacksonian type of convulsion. The patient shown in Figures 1 to 4 had a frontal defect, yet his chief symptom was general convulsions. These were probably in the nature of a defense mechanism as the attacks ceased after the closure of the defect.

Besides the symptoms and discomforts that may be complained of, the patient may also suffer embarrassment from disfigurement when the defect lies over the frontal or temporal areas.

The closure of a cranial defect is indicated in an adult who shows either physical or neurotic symptoms attributable



Fig. 4. *X*-ray of skull on dismissal.

to this defect, who is a fair operative risk, and who is free from acute infections or other defects that might interfere with the normal repair of tissues. The cranioplasty must not be done until the injury to the scalp has entirely healed. Discharging sinuses leading to pieces of necrotic bone call for a postponement of the plastic operation until all dead and infected bone has been removed and the sinus healed. Even an area of granulation tissue must be allowed to heal and become entirely covered with healthy epithelium.

Operations for the closure of skull defects are not only useful in cases where bone has been lost in compound fractures, but also in those cases in which portions of the skull have been removed on account of disease or neoplasm.

Defects of the skull in children do not require operative repair as young patients spontaneously generate new bone to cover their defects in the course of three to six months. Most of this new bone probably comes from the pericranium but I have seen patients who have undergone com-

plete regeneration of new bone who had lost a portion of their pericranium at the time of the injury. The tendency to spontaneous repair generally ceases with the sixteenth to seventeenth year of age.

Of all the various types of cranioplastic operations devised there are only two that are of practical value. In both of these operations a periosteal bone graft is used to cover the defect. The first type devised by Frazier[1,4] places the graft with the bone next to the dura. This makes a very neat closure, but it has one weak point, that is the bone is adjacent to the dura which is comparatively avascular. In the second type, devised by Bagley[2,3] the graft is reversed so that its bony portion lies next to the under surface of the vascular scalp. This second type does not lend itself to so neat a closure, but for practical purposes it is very satisfactory. I have always preferred Frazier's method, but in two cases there later occurred absorption in a part of the graft, followed by a return of symptoms. A second operation was then performed after the method of Bagley which resulted in a solid closure.

* Illustrations showing steps in operations were made from models.

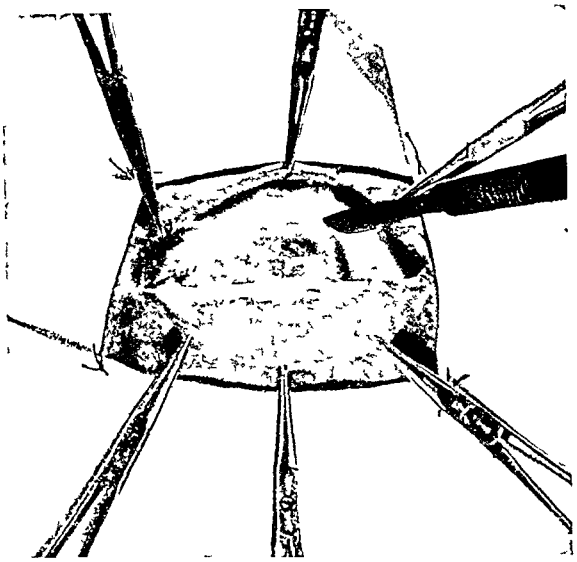


Fig. 5. Scalp flap turned back. Bone defect covered with pericranium exposed. Knife indicates point of incision in pericranium.*

Regardless of the type of operation to be done, the first step is to dissect away any thin scar tissue that might be present over the defect. If this scar is thick and apparently vascular it may be left as part of the scalp flap, but when the scar is very thin and adherent to the dura it must be completely excised. It is often difficult to separate this epithelized scar from the dura as planes of cleavage are generally obliterated. The next step is to make incisions in the normal scalp radiating from this denuded area in such a way as to expose the bone defect and at the same time plan for the closure of the scalp defect. These scalp incisions will vary with individual cases. The chief point to be kept in mind is that the graft must be covered with vascular scalp sutured without tension. In some cases in order to prevent this suture tension the scalp will have to be incised similar to a sliding graft leaving a denuded area of skull in another region of the head. This denuded area can either at the same time or later be covered with a Thiersch or other form of skin graft. In case the scar tissue about the defect is vascular and not too thin to interfere with the viability of the scalp flap a simple

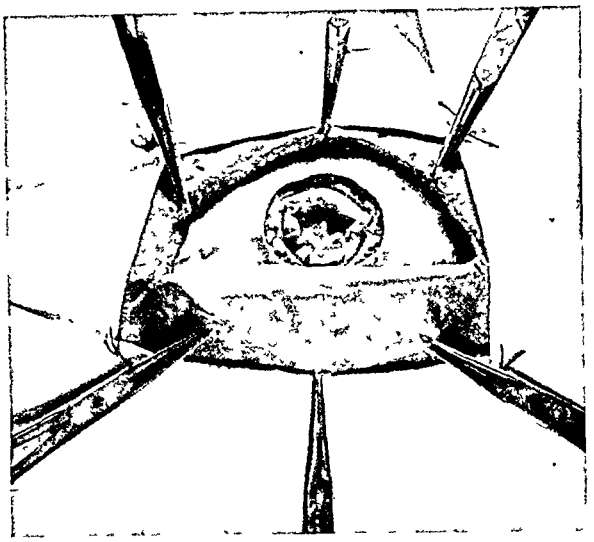


Fig 6. Pericranium elevated and defect prepared for graft.

semi-elliptical incision may be used extending to the pericranium (Fig. 5).

If the Frazier type of repair is to be done

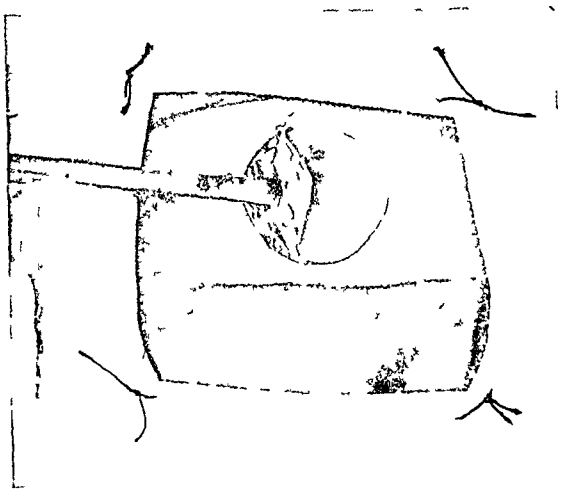


Fig 7. Cutting pericranial bone graft.

the following steps are carried out: The scalp is incised and reflected to expose the pericranium covering the defect. With the point of a knife an incision is made in the pericranium surrounding the defect, about ⅛ inch outside the edge of the bony defect (Fig. 5). With a small sharp periosteal elevator the inner edge of the periosteal incision is separated from the edge of the bone as far as the inner surface of the skull. As a rule this can be done without tearing or opening the dura. If such an opening is accidently made no harm is likely to occur. Some advocate opening the dura and exploring the underlying brain but I see no reason for this unless one suspects an underlying cyst, loose fragments of bone or foreign bodies. After the pericranium is completely separated from the edge of the bone defect, this redundant membrane is folded in over the defect to help fill part of the dead space (Fig. 6). The outer edge of the cut pericranium is then elevated just enough to allow the introduction of sutures. Next, the edge of the bone defect should be examined for rough places or spicules extending towards the brain. These, if present, should be separated from the dura and removed with a rongeur. The outer edge of the bone defect is then freshened and beveled with a chisel or gouge. The defect is now prepared to receive the graft.

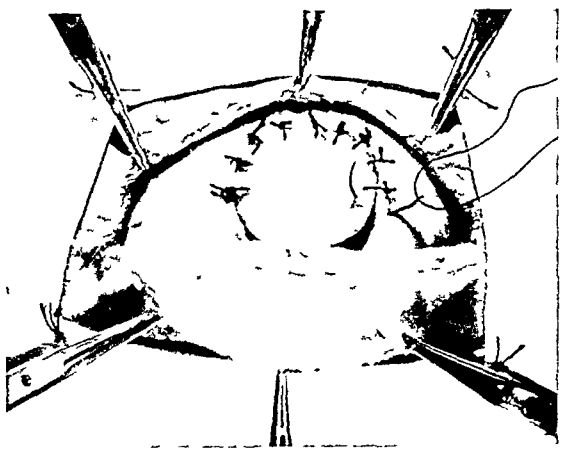


Fig. 8. Suturing graft in place.

The periosteal bone graft is best obtained from the parietal region, but other areas of the skull may be utilized if the circumstances make this more practical. If the defect is small the simplest procedure may be to extend the original scalp incision and to obtain the graft adjacent to the defect. If the defect is large it is generally best to expose the parietal region of the opposite side, making a semicircular incision in the scalp extending to the pericranium. To determine the size and shape of the graft a piece of thin sheet tin or lead is used. This must be thin enough to be very pliable. It is held over the defect and pressed down so that an imprint of the edges of the defect are obtained. With scissors the metal is trimmed about ⅛ inch outside the imprint of the defect. This metal pattern is then firmly held against the exposed area of skull from which the graft is to be taken, and with the point of a knife the pericranium is incised corresponding to the edge of the pattern. The pattern is then discarded. Through the circular incision in the pericranium a small chisel is inserted and the skull cut to the depth of about 1/16 inch, in order to outline the bony part of the graft on the bone. The next step is to use a sharp chisel with a ¼ inch cutting edge to obtain the bone

portion of the graft. This chisel is held in a very oblique position and starting at one edge of the circular cut in the bone the chisel is slowly and carefully driven under the disc of pericranium, chipping flakes of bone from the skull. These chips will vary in size and thickness. It is best to confine the bone chipping to the outer table for if the chisel penetrates the diploe it tends to go too deep and cuts chips too thick to be properly moulded. There is always a tendency for the chisel to go too deep and as soon as this is noted it should be withdrawn, the handle lowered, and chipping on a higher level resumed. If this chipping is properly done the chips of bone will remain attached to the under surface of the pericranium (Fig. 7). As the cutting of the bone proceeds the graft tends to curl upward. After it is entirely cut the graft can be moulded between the thumb and fingers to give it its normal contour.

The next step is to place the graft over the defect, the bone side next to the dura. The edge of the periosteum of the graft is then sutured to the edge of the pericranium about the defect by fine silk, interrupted sutures (Fig. 8). The scalp wound is then closed by two rows of interrupted sutures, deep and superficial. Fine black silk is very satisfactory. No drainage is required. If there is persistent bleeding from the skull where the graft was removed, this can be controlled by bone wax.

The second method of closure is by means of a hinged graft. In this method the graft is cut from the area adjacent to the defect, the pericranium being left uncut along the border where the edge of the graft and the defect meet (Fig. 9). The periosteal bone graft is cut as in the first method leaving a hinge of periosteum on one side. The graft is then folded over the defect, the bone chips being on the outer side. The pericranium of the graft is then sutured to the pericranium about the defect with interrupted silk sutures (Fig. 10). The scalp is then closed in the usual manner.

In order to make a neat closure with the hinge graft it is necessary that one side of the defect be in the form of a straight edge, otherwise it will be impossible to obtain a perfect pericranial hinge. The first time I attempted this method was in a patient with a circular defect; who, after a cranioplasty by the first method, showed a partial absorption of bone in the graft with a return of symptoms. A second operation was made by the hinge graft and when completed looked far from neat; nevertheless, the final result was a firm closure with a disappearance of symptoms. Confronted again with a similar case of

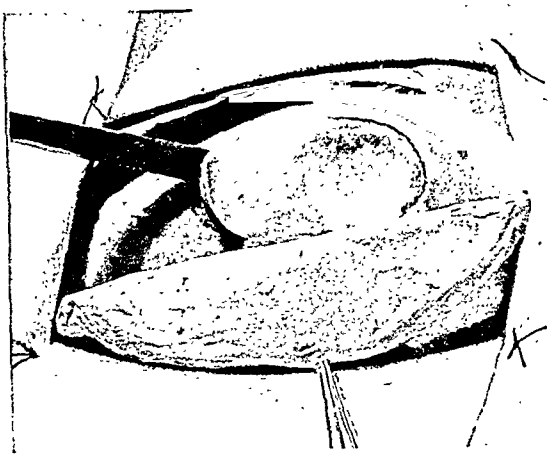


Fig. 9. Preparing graft in hinge method.

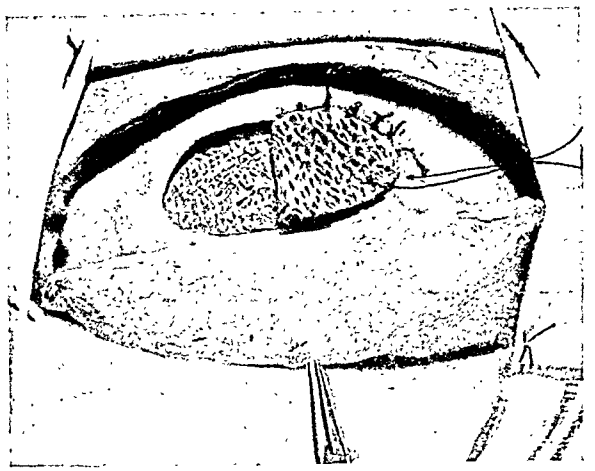


Fig. 10. Suturing hinge graft in place over defect.

absorption of the graft I would dispense with the hinge, cut the graft completely free from the skull as in the first method,

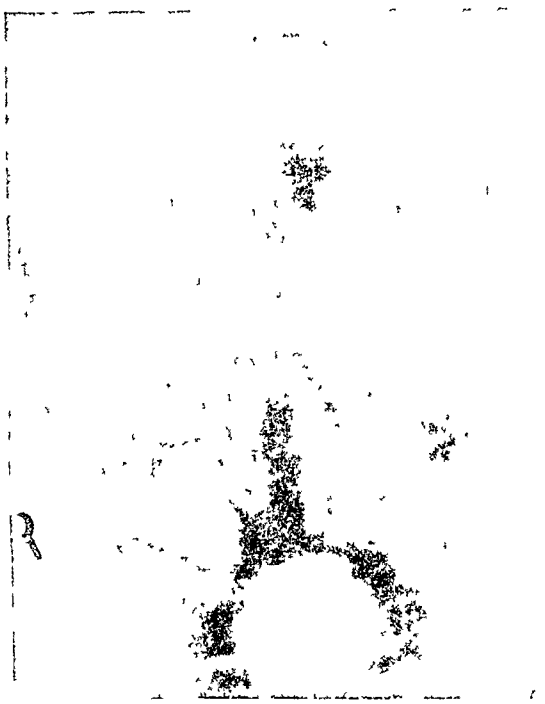


Fig 11 Case B. C. Anterior-posterior view of defect on first admission

and then suture the graft over the defect with the bone side out.

In the case cited here the hinge graft was of distinct advantage. In this case of the repair of a very large cranial defect there was an absorption of one side only of the graft. The edge of this secondary defect was almost straight which made it very easy to cut a long narrow strip of graft and turn it over the softened area.

There is a marked similarity in the history, complaints, and postoperative course of patients suffering from cranial defects. The latter case, however, showed some unusual features that warrants its report in detail:

Case 1. B. C., white, male, aged twenty-one, was admitted to the Methodist Hospital, Indianapolis, on May 30, 1928, complaining of left-sided epileptic fits and right-sided cranial defect.

The past history showed that five years before the boy was in a stone quarry accident when a large stone crushed the right side of the skull. The treatment at that time was the removal of fragments of skull through the scalp wound followed by closure of the scalp. Aside from some weakness in the left hand and a certain amount of fear on account of the large skull defect he considered himself recovered. Three years later he began to have twitchings in the left hand which gradually

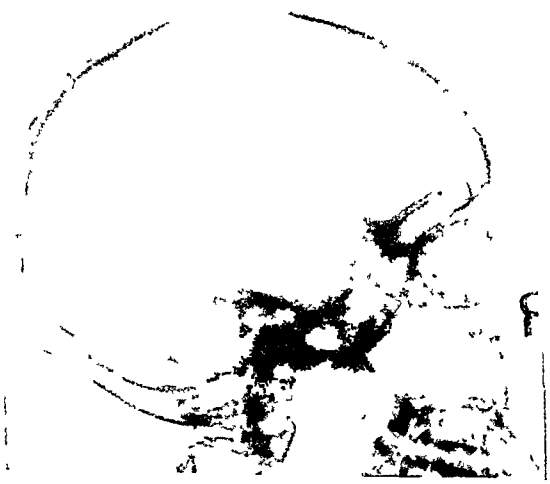


Fig. 12. Case B. C. Lateral view of defect on admission.



Fig 13. Case B. C Second admission showing partial absorption of graft.

extended to the entire arm. This was soon followed by a similar twitching in the left leg and foot. The left hand then became spastic and paralyzed and he began to have typical left-sided Jacksonian epilepsy. These attacks

gradually increased in frequency, and during the past three months before admission were occurring at intervals of fifteen to thirty minutes. He rarely lost consciousness during these attacks.

The remaining personal history was unimportant, and the family history was negative except that the mother was confined to bed with an active pulmonary tuberculosis.

Examination showed a well-nourished young man of apparently good intelligence. Over the right parietal area was the largest skull defect that I had ever seen in a living person (Figs. 11 and 12). When the head was raised there was a very marked depression, and when the head was lowered there was a marked bulging showing that the brain underwent considerable movement on changes in posture. Most of the scalp over the defect was replaced by very thin scar tissue. The patient suffered attacks of Jacksonian epilepsy involving all of the left side from the feet to the neck muscles. These attacks occurred at intervals of about every fifteen minutes. All drugs failed to modify these attacks to any noticeable degree. The left leg was spastic but not paralyzed. The left hand was paralyzed and held in the typical "claw hand" position. Further examination was essentially negative.

Operation: On June 6, a cranioplasty was performed after the method of Frazier. The graft was obtained from the opposite parietal region. With difficulty the graft was cut in one piece. The operation consumed four hours and the closure was satisfactory.

Subsequent History: The patient had one mild convulsive attack the day following the operation. This was his last and only epileptic attack for two and one-half years. The wounds healed per primam. He was discharged July 6. He walked out with a slight limp due to the spastic left leg. No improvement was noted in the left arm or hand. The cranial defect was perfectly solid to the touch.

About one year later he reported that he was married and making his living doing general work on a farm.

Second Admission: On December 13, 1930 he was admitted to the Robert Long Hospital, Indianapolis, complaining of a slight return of the Jacksonian attacks, and a softened area at the upper edge of the old defect (Fig. 13). Examination showed that there had been some absorption of the edge of the upper part of the graft about ½ inch wide through which pulsation could be felt. The remainder of the graft seemed perfectly solid.

Operation consisted in exposing the softened area only and covering this with a hinged graft which seemed ideal in this case. Wound healed per primam and all suggestion of epileptic attacks ceased. The patient was discharged four weeks after his admission apparently in good condition. While in the hospital he showed an elevation of temperature of about one degree daily. Tuberculosis was suspected as he had been exposed to this infection while living with his mother who had recently died, but examination failed to demonstrate this condition.

Third Admission: February 15, 1931. Four weeks after his last dismissal he was re-admitted to the Robert Long Hospital complaining of diarrhea and abdominal cramps. No history of epileptic attacks.

Examination showed the cranial defect firmly closed. General examination showed a marked degree of diffuse acute pulmonary tuberculosis together with acute tuberculous enteritis. He was discharged from the hospital March 9, 1931, unimproved. He died about six weeks later.

Comment: This case shows some interesting features. It is unusual to see such an extensive cranial defect, and it shows that there is probably no defect too large to attempt a closure. If a first attempt is not successful, repeated attempts will finally produce a solid skull.

In the past twenty-five years I have made many attempts to relieve patients with Jacksonian epilepsy using all methods described. With the exception of slight or temporary improvement all of these cases resulted in failure. In the foregoing case, the stopping of the attacks following the stabilizing of the brain within the skull by closing the defect makes me feel that the cause of the attacks may be due more to the motions of the brain from this instability than to the adhesions between the brain and dura.

[For References see p.295.]

OCCUPATIONAL THERAPY IN THE TREATMENT OF FRACTURES*

J. WILLIAM HINTON, M.D., F.A.C.S.

NEW YORK CITY

THE results from the treatment of different types of fractures that involve the joints have not been uniformly good; if the results were satisfactory there would not be such a diversity of opinion as to the method of treatment to be employed. I have had the opportunity to treat and observe a very large and varied group of fractures on the Fourth Surgical Division of Bellevue Hospital for the past eight years and the general conclusion one must draw is that the main objective is not necessarily anatomical alignment but rather the functional result and economic restoration. Unfortunately the tendency has been, due to the routine use of roentgen ray, to over-emphasize the anatomical result and minimize the earning capacity and functional result. It does not necessarily follow because the patient has a good anatomical alignment of the fracture that he will have a good functional result, and with the alignment not so perfect, but with a different type of treatment a much better functional result may be obtained and the individual restored to earning capacity much sooner.



FIG. 1. Patient filing wood to obtain wrist motion



FIG. 2. Patient sawing which gives wrist, elbow and shoulder motion.

In this discussion it is not intended to advocate any particular type of treatment, as it is obvious that all types of treatment have a selected field of usefulness, and Dr. Carl G. Burdick, Director of the

* Read before the Medical Association of Greater New York, December 21, 1931.



FIG. 3. Patient weaving, giving flexion of elbow or knee.



FIG. 4. Patient is using a brace and bit for rotation of shoulder or pronation and supination of forearm.



FIG. 5. Patient weaving on circular frame for abduction and rotation of shoulder.



FIG. 6. Patient using a treadle saw for flexion and extension of ankle.

Fourth Surgical Division of Bellevue Hospital, not being satisfied with the results from the treatment of fractures, established a follow-up clinic in November, 1925 to evaluate the results from the different methods employed. After observing these patients for one year in the follow-up clinic it was felt that the results were not sufficiently satisfactory and the poor results seemed to result more from delay in treatment than from the type of treatment employed, so Dr. Burdick decided to have all fractures, regardless of type, treated as emergencies and immediately reduced, and at that time Dr. Lewis and I were assigned to the care of all fractures on the division; this routine was carried out for a two-year period. It was noted that by reducing fractures immediately after admission some of the problems previously encountered in difficult cases were simplified.

During the past six years 1725 fractures have been treated on the Fourth Surgical Division. The majority of the joint fractures have been treated by immediate reduction with the application of a cast, or moulded splint, and the one exception has been fractures of the shoulder which have been treated by traction. It has been most unusual to have to resort to an open operation in any of the fractures, provided these patients upon admission were immediately *x*-rayed, and treatment instituted. One should remember that there is no 100 per cent satisfactory method of treatment to be employed in all fractures of a similar type, but the treatment has to be selected to fit the given case and if these cases are seen early and treatment instituted immediately, the difficult ones are made easier, while if treatment is delayed easy ones are frequently difficult.

The after-treatment of fractures is most important so as to shorten the disability and give a good functional result. During the first year the follow-up clinic was run different types of physiotherapy were used, chiefly baking and massage, with a moderate degree of success, but particularly in the joint fractures the results did not seem to be as satisfactory as one desired, and it was then thought advisable to institute occupational therapy in all types



FIG. 7. Patient weaving to obtain abduction of hip.

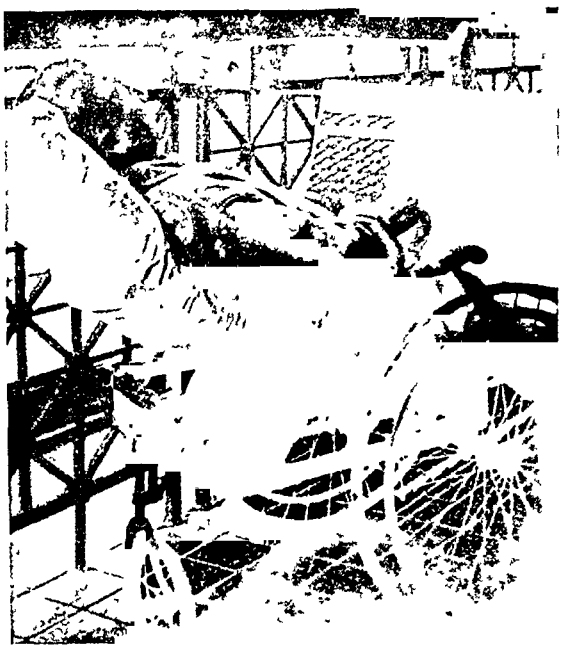


FIG. 8. Patient doing cord knotting for flexion of hip.

of fractures that involved the joints, and with the cooperation of Miss Merritt, Director of the Occupational Therapy Department, the joint fractures have been referred to her since that time with most encouraging results. A detailed report[1] as to the methods used was made before the American Occupational Therapy Association in 1929 and those interested in the exact technique of treatment are referred to this work. All told there have been 1143 cases in the Occupational Therapy Department from the Fourth Surgical Division, which include 1101 fractures, and most of these have involved the joints, or were closely associated to a joint; there were 42 dislocations in the foregoing group. Miss Merritt feels that the results from occupational therapy alone have been better than in those cases in which the patient has received both physiotherapy and occupational therapy. The patients from the Fourth Surgical Division have received only occupational therapy, but some of the other divisions have employed physiotherapy and occupational therapy on alternating days which has given Miss Merritt an opportunity for her observations.

The illustrations represent some of the work done by patients with fractures involving the joints, and one can see the interest that the patient has in his work. Of course, there are three work periods, alternating with rest periods, and the work periods being two to three minutes and alternating with one to two minutes of rest. As the patient's condition progresses the work periods are increased and the rest periods shortened, and the type of work is changed so as to require more energy.

There are two reasons for selecting occupational therapy in preference to massage and other types of physiotherapy: The first is reestablishing confidence in the patient, so that he will use the injured part. It then soon becomes a competitive task and one tries to do as well, if not better than someone else with a similar injury doing the same work. Secondly, it is known that vigorous massage, in children, may produce myositis ossificans, particularly in elbow fractures. This has been demonstrated with roentgenologic findings. If this can happen in children it would seem that it might happen in adults to a lesser degree, and instead of the patient being benefited by prolonged massage his progress may actually be retarded.

If one observes the interest that patients put into their work, at the same time completely forgetting their injury, the benefit derived from this type of treatment in contrast to physiotherapy is obvious.

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* Continued from p. 291.

ADVANTAGES OF URETERO-INTESTINAL ANASTOMOSIS FOLLOWING NEPHRECTOMY FOR TUBERCULOSIS IN CERTAIN CASES*

A. R. STEVENS, M.D., F.A.C.S.

NEW YORK CITY

I AM encouraged to draw attention to the advantages of ureteral transplantation into the bowel in dealing with certain patients who still have an intolerable urinary condition after nephrectomy for tuberculosis, because of my gratifying experience with two such personal cases so treated. Both patients were afforded immediate relief and at the end of two and one years respectively are at work, entirely comfortable and in excellent general condition.

CASE 1.* An Italian girl, aged fifteen, was first admitted to the hospital March 5, 1926, complaining of pain in the back and right lower abdomen of four months' duration, but especially of marked frequency of urination which had become progressively worse the past eight months. Fortunately the child was a sound sleeper and the night frequency was practically incontinence. Hematuria had been noted for one week.

Examination: The patient was in fair weight and did not appear very sick. General examination, including lungs, was quite negative. There was slight tenderness in both costovertebral angles, more on the right side; the kidneys were not palpable. The urine was turbid, contained some albumin and considerable pus and blood; but no tubercle bacilli were found. Hemaglobin, 75 per cent; leucocytes, 8000.

Cystoscopy: Bladder capacity was only 1½ ounces. Urine very cloudy. Bladder showed marked general inflammation and bled easily. After several abortive attempts, the right ureter was finally catheterized and very turbid urine was obtained; this contained much pus but no tubercle bacilli were found in the stained slide; the urea percentage was 0.3, and indigocarmine given intravenously appeared in a trace only, after twenty minutes. The left ureter was not catheterized, but urine obtained from the bladder after thorough irrigation contained no pus; its urea percentage was 0.5 and indigocarmine appeared in seven minutes in deep concentration. A right pyelogram showed a non-rotated kidney with dilated calyces, some with an irregular contour indicating a destructive process.

Operation: On March 29, 1926, right nephrectomy under general anesthesia was done. The ureter was very indurated and nearly 2 cm. in diameter. It was cut across near the pelvis, as I thought the patient's condition did not permit complete ureterectomy. The kidney was of normal size, and contained many small abscesses; the histological report was tuberculosis.

On April 22, phenolsulphonephthalein output (after intramuscular injection) was 15 and 10 per cent for the first two hours. Operative recovery was satisfactory but the urinary frequency continued. Cystoscopy revealed general cystitis and ulcerations about the right ureteral orifice. Two applications of 20 per cent acid nitrate of mercury were made to these ulcers. Patient was discharged from hospital May 22.

She was re-admitted October 6, 1926, for five weeks, as the urinary frequency had become even more marked, often every five to ten minutes, and there was occasional hematuria. Cystoscopy showed tubercles about the right ureteral orifice and general cystitis. The left ureter was catheterized and clear urine obtained; microscopically there were only a few scattered leucocytes and no tubercle bacilli. The patient was discharged to a tuberculosis sanatorium.

*This is also Case 1 in article entitled "Pyoureter" by H. S. Jeck.[1]

* From the Urological Department, Bellevue Hospital. Read before the American Association of Genito-Urinary Surgeons, Buck Hill Falls, Pa., June, 1931.

She was re-admitted February 6, 1927, with no improvement of the urinary status. The cystoscopic picture was about the same

Patient was discharged March 27 to a sanatorium for tuberculosis. Bladder irrigations of some sort were tried with no relief.



Fig. 1. Case I. Intravenous uroselectan urogram, thirteen months after implantation of left ureter in large bowel. Operation was performed three years after right nephrectomy for tuberculosis.



Fig. 2. Case II. Intravenous uroselectan urogram, made three weeks after implantation of right ureter in large bowel. Operation was performed three years after left nephrectomy for tuberculosis.

and the bladder capacity was ½ oz. Remembering the condition of the ureter as found at the first operation, I suspected that this might be a fundamental cause of trouble, and on February 24, *removed the remainder of the right ureter* down to the bladder, easily drawing it under the broad ligament. I was amazed to find that the ureter had changed markedly in size and consistence; it was soft and pliable and no larger than a lead pencil. During this procedure the peritoneal cavity was entered, several ounces of clear fluid evacuated, and several loops of bowel studded with tubercles were noted. The bladder was palpated, was small and its wall seemed to be 7 or 8 mm. thick. The ureter showed microscopic evidence of tuberculosis. The convalescence was uneventful but the urinary condition was not helped by this operation.

Cystoscopy on May 16, 1927 showed general cystitis with some small ulcers on the left side of the bladder; two typical tubercles were seen. The bladder capacity was still small. Ureteral transplantation was considered at this time, but fearing extensive intraperitoneal adhesions, I advised further waiting.

In December, 1928, the patient was virtually incontinent day and night. Fortunately this did not seem to interfere with sleep and she held her weight. The bladder capacity was 1½ ounces and cystoscopy showed some improvement in the appearance of the mucosa in that there were no ulcerations or tubercles. Indigocarmine given intravenously appeared in ten minutes distinctly but not of deep color.

The patient's fifth admission to the hospital was on May 21, 1929. The situation had been

carefully weighed. The girl was one of eight children living with the two parents in a four room flat. Her bed was soaked with urine every night and pads were worn by day and she was unable to mingle with friends. She and her mother were quite willing to take any risk involved in a procedure which offered hope of relief.

On May 24, 1929, *the left ureter was transplanted into the upper rectum* through a left rectus incision extending from the umbilicus to the pubis. The rectum had been thoroughly irrigated just prior to operation. The work was begun under spinal anesthesia but general anesthesia was required later. On opening the peritoneal cavity, it was a great relief to find but few adhesions and no tubercles present. There was no evidence of tuberculosis of the fallopian tubes. The ureter was located at the junction of the internal and external iliac arteries and followed to the bladder. The overlying peritoneum was incised and the ureter cut across near the bladder with a carbolized knife; the lower end was tied and the upper clamped. The ureter was uniformly dilated, about 8 mm. in diameter. The small intestines were held in the upper abdomen by gauze pads. The anastomosis of ureter with large bowel followed closely the earlier technique of Coffey, no catheter being used in the ureter. The musculature of the bowel was dissected well back from the mucosa, affording ample space for the large ureter. This was held in place by the suture in the end of the ureter, brought out through the bowel wall about 1.5 cm. below the opening made in the mucosa to admit the ureter; its position was further fixed by including a little of the ureteral wall in the lowest of the five Lembert sutures of fine chromic gut closing the muscle and serosa over the ureter. A few reinforcing sutures were employed and the site of the fixation suture from the end of the ureter was similarly covered. The posterior peritoneum was carefully sutured and the abdominal wall closed in the usual manner, with a cigarette drain to the site of anastomosis. Care was employed that the ureter should follow an easy course to the bowel without angulation or any tension. A tube was left in the rectum four or five days after operation.

The ureter seemed dilated with urine at the end of the operation. I feared the closure of the bowel over the ureter may have been made too tight, and was prepared to do nephrostomy later. However, urine came from the rectum during the night after operation and 11 ounces were recovered the next day, and 40 ounces during the succeeding twenty-four hours. The temperature touched 102°F. the first postoperative day, but subsequently did not rise over 100°F. At no time was there any tenderness over the kidney. The patient made an excellent recovery and was discharged from the hospital nineteen days after operation with the wound healed. She had good rectal control, holding urine two to three hours and not wetting the bed at night.

Two months after operation the intramuscular phthalein test was 10 and 5 per cent, collections being made at the end of one hour and ten minutes and two hours and ten minutes. Six months after operation the phthalein test was the same, and the patient had gained 15 pounds.

A year after operation she looked and felt very well. Bowels usually moved easily, but patient occasionally took a little mineral oil. She rarely got up at night to urinate and held urine from two to four hours by day. There was no wetting of bed or clothes. Intravenous uroselectan urograms were made (Fig. 1) and show some dilatation of the ureter, probably more than existed at the time of operation. Eighteen months after operation, the phthalein output was 7 and 20 per cent for two hours.

Two years after operation (May 17, 1931) the patient was cheerful and doing daily clerical work. She had no complaints and was urinating every two to three hours when standing but held urine four hours when sitting. She frequently sleeps all night without interruption and never gets up to urinate more than once. When exercising strenuously she occasionally leaks a little urine, otherwise the control is perfect. Intravenous phthalein test shows 5, 10, 10, and 5 per cent for the first four half-hour periods. Intramuscular test was done for comparison with earlier reports; it was 10 and 15 per cent for two one-hour periods. These certainly show no diminishing renal function, but rather an improvement since operation.

Case 11. An Italian man, aged thirty-six, was admitted to the hospital March 17, 1930,

complaining of frequency of urination and dysuria. Two years earlier his left kidney had been removed for tuberculosis. But the urinary distress really became worse and for five months after operation there was also occasional hematuria. At time of admission, urination was at intervals varying from fifteen minutes to one hour. There was almost a constant desire to empty the bladder, and the patient frequently bent double with straining and pain during the act.

The family history was negative for tuberculosis, and the patient's own past history was irrelevant except as stated.

Examination: The patient was a small, thin, undernourished man, weighing 97 lb., and appeared worn out from loss of sleep. We could find no evidence of an active tuberculous process anywhere outside the urinary tract. Radiograms of the chest were negative and examination of abdomen and genital tract was likewise negative. The urine showed a trace of albumin and much pus. No tubercle bacilli were found on three microscopic examinations. The Wassermann test was negative. Six blood examinations showed non-protein nitrogen varying between 33 and 42 mg. per 100 c.c. of blood, and creatinine between 1.6 and 2.2. Phenolsulphonephthalein test (intra-muscular injection) before operation, varied between 15 and 30 per cent and 35 and 20 per cent for two hours. This certainly precluded any marked tuberculosis of the remaining kidney.

Cystoscopy: The bladder capacity was not over 2 ounces. There was general cystitis with few small ulcerations on the right posterior wall. The right ureteral orifice was large but would admit no catheter over 2 cm.

The patient was given six weeks' treatment of the bladder, trying irrigations and also phenol instillations. These seemed to lengthen the average intervals between urinations, but the longest was never over one hour. However, the pain continued to be severe. The temperature was elevated at times, but an afebrile period was chosen for operation, after two postponements.

Operation: On April 29, 1930, *transplantation of the right ureter into the upper rectum* was done through a midline incision, under spinal anesthesia. The technique employed was virtually the same as in the first case. The ureter was soon found, and proved to be nearly 2 cm. in diameter. The large bowel was not over 3 cm. in diameter, so that the job looked formidable. However, after the ureter was cut across and allowed to empty, its size diminished markedly. The urine collected was later examined and it contained only an occasional leucocyte; no tubercle bacilli were found. The muscular layer of the bowel wall was dissected widely to make room for the large ureter. Two of the Lembert sutures grasped a small bit of ureteral wall, care being taken to avoid blood vessels. A second layer of Lembert sutures was placed over the first layer and over the ligature in the bowel wall coming from the end of the ureter. Care was exercised that the ureter should follow an easy course without angulation or tension. The posterior peritoneum was carefully sutured, and the abdomen closed without drainage. As in the other case, a tube was left in the rectum for about five days.

Urine came from the rectum the day after operation and there was a good output daily thereafter. Three days after operation the non-protein nitrogen rose to 48, but soon dropped to normal. The temperature reached 103°F. on the third postoperative day, but was not above 100°F. afterwards. In two weeks, the patient was passing urine every two or three hours, about 7 ounces at a time. There was no tenderness noted in the kidney region at any time. Urograms were made after giving uroselectan intravenously, nineteen days after operation. These showed dilatation of the ureter (Fig. 2), about as would be expected from the operative findings. Twenty-two days after operation the patient left the hospital, with the wound healed and a gain of 4 lbs. in weight.

Six months later, he weighed 115 lb. (a gain of 18 lb. since operation), had no discomforts and controlled the rectum day and night. He did not drink much water and hence emptied his rectum only four times in twenty-four hours. He would notice a little pain for a few minutes occasionally, in the right costovertebral angle, "on cloudy days." A year after operation (May 17, 1931), he reported that he could sleep continuously for seven to eight hours, and urinated by day only once in five or six hours, always having a good control. He was feeling well and working everyday as a clerk. His weight was 116 lbs.

Phenolsulphonephthalein test (intravenous) was 5, 10, 5, and 10 per cent for the four succeeding half-hour intervals.

It is a matter of common knowledge among urologists that the change in urinary symptoms following nephrectomy for tuberculosis cannot be predicted. If frequency has been of short duration, it may cease quickly. But with a long history and tuberculous involvement of the bladder, no improvement may be noted during the stay in the hospital. In some instances, with proper and prolonged hygienic care including heliotherapy, the patient may become entirely comfortable in time. Indeed, such care should be urged upon all patients following any surgical operation for tuberculosis. Those individuals with continued bladder symptoms after nephrectomy should also be given the benefit of instillations: bichloride of mercury, phenol, medicated oils like gomonol, and methylene blue all have their advocates. If ulcerations exist, treatment with fulguration or caustics is often useful. Urinary sumptoms may be prolonged because of the persistence of a secondary infection or the co-existence of lesions of the ureter or prostate and seminal vesicles. When all of these resources have been exhausted, distressing symptoms may still remain. To this unhappy and pitiable group of patients with frequent and painful urination, life is scarcely worth while. They will obtain relief only through some operative procedure aimed at sidetracking the urine and thus putting the bladder at rest. Suprapubic drainage gives temporary relief only, if at all. Nephrostomy seems justifiable only as a preliminary measure to improve the kidney function and reduce the degree of hydronephrosis preliminary to diversion of the urinary stream by some operation on the ureter. Ultimately, implantation of the ureter to the skin or in the intestinal tract seem to offer the only possibilities of relief.

The decision between these two courses depends first on a comparison of the two operations for diverting the urinary stream, as done for any and many reasons; and secondly, on a consideration of what is best for tuberculosis cases in general and for a specific patient in particular.

The skin operation was decidedly the less risky until recent years when rapid strides have been made in improving the technique of anastomosis of the ureter and the bowel. Supported by much experimental work, clinicians have been able to report excellent results. Should the immediate mortality from this operation be shown in the future to be but little greater than that to be expected from implantation of the ureter in the skin, and the expectancy of life as favorable (and these are possibilities), there is no question that the bowel operation will become the one of choice in most cases. The absence of urinous odors about the individual and of an apparatus needing continuous care, together with the greater comfort and greater freedom of action following the bowel operation, are the chief arguments coming to mind.

At present, in the hands of most surgeons, anastomosis of the ureter to the skin has less immediate mortality than anastomosis to the bowel, and is undoubtedly the method of choice in debilitated individuals. Papin[2] reported that a search of the literature to 1925 showed 50 per cent mortality for all ureteral transplants to the large bowel. Yet skilled surgeons with large opportunities for this type of work, such as Coffey, C. H. Mayo and Lower, have shown what may be expected of the newer methods of ureterointestinal anastomosis. Coffey's report[3] of 20 consecutive operative cases, many with double implants at one operation, with only one hospital death, is particularly encouraging.

The late results of transplantation of the ureter to either skin or bowel seem to be largely dependent on the presence or absence of obstruction and infection. Both may be encountered after either type of operation. Obstruction following anasto-

mosis to the skin is controllable by the use of a permanent ureteral catheter or regular dilatation or incision, whereas obstruction at the end of a ureter implanted in the bowel is out of reach, dependence being entirely on the development and execution of good technique. One naturally supposes that renal infection would more frequently follow implantation of the ureter in the bacteria-infected gut than in the relatively clean skin. Yet only experience will answer this question. McKenna[16] has shown an absence of infection and no decrease of function in the kidneys of experimental animals, more than a year after the ureter was transplanted into the bowel by Coffey's method. Also let us not forget the natural history of exstrophy cases,[4] that over 50 per cent of these patients die under ten years and but 30 per cent reach the twentieth year; and in a large percentage of these deaths infection is the cause.

While the length of life following transplantation of the ureter to the skin is presumably as favorable as that following transplantation to the bowel, I have not encountered, in a perusal of recent publications, a patient who had lived more than six years after the skin implantation, at the time of the report. No attempt has been made as yet to follow up this point in the earlier literature. On the other hand, Turner[5] reported patients living up to fifteen years following ureteral transplantations to the bowel after the manner of Stiles (no attempt to construct a mucous membrane valve); and I know of two patients who lived at least seventeen years, one after a Maydl[4] and one after a Bergenhem[6] operation for exstrophy. Ester[19] reported the results of a case, twenty-four years after such operation.

It seems to be generally conceded, and is supported by much experimental work, notably that of Sweet and Stewart,[7] that the lymphatics of the ureter represent the chief route followed by infections from the bowel to the kidney, especially in the early portion of the trip; and operations have been devised to protect the end of the ureter from bowel contamination. It would seem that sealing by mild cautery coagulation of the cut end of the ureter should serve this purpose. But I have not yet tried this technique. Coffey and others have advocated the use of an indwelling ureteral catheter during the early days after operation, believing that fewer kidney infections follow and that temporary obstruction by edema is thereby avoided. The ligature holding the catheter in the ureter probably closes the ureteral lymphatics to infection temporarily, and some sealing of the free end of the ureter left after the sloughing distal to the ligature is caused by the traumatic pressure of the ligature. On general principles, I have not liked this use of catheters in the ureter as the presence of a foreign body in any part of the urinary tract promotes infection. But many operators have followed Coffey's lead in this matter, and the experimental work of Sisk, Wear and O'Brien[18] adds evidence in favor of the catheter. Moreover, I have had no trouble with temporary obstruction after operation, in a small experience. Five ureters have been implanted into the bowel by the Coffey method in four patients, and urine has appeared in the rectum in all cases within twelve or fourteen hours, and an abundant amount expelled in the next twenty-four hours. In only one case was a catheter used in the ureter, and curiously the corresponding kidney was the only one in the series which was tender subsequent to operation. C. H. Mayo[8] reported in February, 1930, that he still used no catheter in doing uretero-intestinal anastomosis, but operates on but one ureter at a time.

As for the results in tuberculosis cases in the recent literature, Rosenkranz[9,10] reported two patients alive and well two and four years after ureteral transplantation in the skin (done subsequent to nephrectomy for tuberculosis); and one that died in two weeks. He felt the latter represented an error of judgment in that

the patient was weak and probably had abscesses in his kidney. Mathé[11] had two similar cases, one traced for seven months. Keyes[12] transplanted a single ureter to the skin in two patients who had previously undergone nephrectomy, and they were comfortable four and five years later; and both ureters in one patient of bilateral renal tuberculosis, known to be living two years later. Papin[13] reported 13 such transplants of ureters in tuberculosis, one apparently well six years later.

Hinman[14] transplanted a ureter to the bowel in four patients subsequent to nephrectomy for tuberculosis. One died four and a half years later from anuria and toxemia due to obstruction by a calculus in the upper ureter; one died a year later from pneumonia; another was living two years and two months after operation; and the fourth died twenty-three days after operation from miliary tuberculosis. Coffey[3] included in his report of November, 1929, two similar patients, living a few months after operation. Kinloch and Drexler[15] published one similar patient living six months after operation. Burnell[20] reported one patient, able to work, with occasional renal pain, one year after operation. Walters and Wright[17] included in a report of operations on single kidneys and ureters, two cases of ureteral transplantation to the bowel subsequent to nephrectomy of the other side for tuberculosis; both of these patients died; they were described as having had "irreparable" disease of the kidneys. Evidently some operations in both groups have been undertaken with misgivings in very sick patients, with the hope of relieving an intolerable condition.

It seems clear that at the present time there is not sufficient evidence available to permit a final conclusion concerning the immediate risks and ultimate prospects of life following the implantation of a single ureter to the skin or in the bowel. But the degree of comfort experienced after operation is certainly in favor of the latter procedure.

My present attitude is that all patients who still have urinary distress after nephrectomy for tuberculosis should have both general and local treatment for a long time before the surgeon is justified in concluding that nature will not afford relief. But when that decision is reached, some type of exclusion of the bladder must be considered. Debilitated persons and those whose remaining kidney is known to be tuberculous, should be offered transplantation of the ureter to the skin of the iliac region. I should not feel justified in transplanting to the bowel a ureter which is discharging tubercle bacilli. Those patients in better general condition whose kidney has fair function and cannot be proved tuberculous, will derive much more comfort and greater satisfaction from uretero-intestinal anastomosis.

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[For Remainder of References see p. 284.]

ABERRANT ENDOMETRIUM*

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WHEN tissue resembling the true endometrium or endosalpinx is found elsewhere than along the lining of the lumen of the uterine cavity or Fallopian tubes it is called aberrant endometrium. The pathological process has been termed endometriosis or adenomyosis and the individual lesions have been named endometriomata, endometrial transplants or implants, hemorrhagic perforating cysts and chocolate cysts of Sampson. The adenomyomata are those tumors that contain smooth muscle in addition to the endometrium-like glands and stroma. They have also been called, when in the Fallopian tube, adenomyosalpingitis, adenomyositis tubae and salpingitis isthmica nodosa.

HISTORY

Adenomyomata of the uterus have been known for a long time. According to Graves[1] our earliest knowledge of endometriosis dates back to 1860 when von Rokitansky first described an adenomyoma as a pathological entity. Shroeder, Herr and Gresskoff, according to Breus and Cullen,[2] were able to collect a total of one hundred cases up to 1884. Two years later von Recklinghausen again described these adenomyomata and suggested that they arose from a rest of the Wolffian body. Cullen[3] was the first definitely to identify endometrial tissue springing from some point outside of the uterus. He reported an adenomyoma of the round ligament in 1896. Two years later, after removing a similar tumor from the round ligament on the opposite side of the same patient, Cullen[4] suggested that the glands might be due to an abnormal embryonic deposit of a portion of Müller's duct. This was suggested, he said, from the striking resemblance of the glands of the adenomyoma to those of the uterine mucosa and from the fact that their stroma resembles that of the uterine mucosa. At this time he referred to a case reported by A. Martin in 1891. The patient was seventy years old and complained of a rapidly growing tumor. The growth sprang from the left round ligament and contained 12 litres of chocolate-colored fluid. There were several small cysts in the pedicle which contained clear fluid. One of these cysts was lined by low cylindrical ciliated epithelium. Quoting from Cullen, "Martin says that in this case the structure and contents corresponded to those of tumors arising from the parovarium."

Shortly after the first report of an adenomyoma originating outside of the uterus, according to Cullen,[4] Pfannenstiel reported adenomyomata in the inguinal region and vagina of a patient, Herff reported two vaginal adenomyomata and Blumer reported an adenomyoma in the inguinal canal.

The first report in the English and American literature of endometrium-like tissue in the ovary was made by Russell[6] in 1899. He refers to a case reported by Burkhard* in 1896. It was not until the third decade of the twentieth century, however, that the frequency of this condition was recognized. In 1917 an article appeared by Emil Novak[9] on hematoma of the ovary, which does not mention aberrant endometrium. While directly preceding this in the same bulletin is an excellent paper on adenomyoma of the rectovaginal septum by Cullen.[14]

Adenomyomata of the umbilicus containing uterine mucosa were first described by Cullen[5] in 1909. In an article on this

* *Virchow's Archiv*, vol. 144.

subject three years later he reported a total of 9 cases.

The first report of a case of adenomyoma of the rectouterine or rectovaginal septum I was able to find in the English or American literature was by Lockyer[7] in 1913. Shortly after this 2 more cases were reported by Cullen.[8]

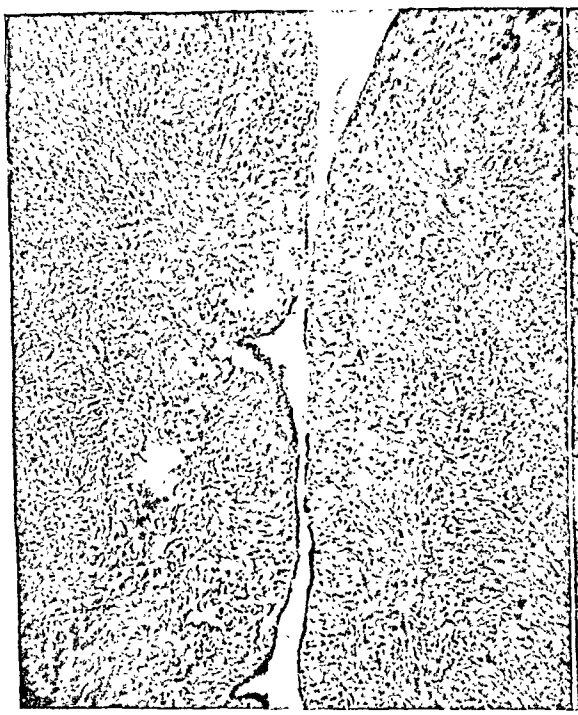


Fig. 1. Invagination and metaplasia of germinal epithelium covering ovary. Note variation in height of epithelium. Surface of ovary is above.



Fig. 2. Greater magnification of an area shown in Figure 1. Note variation in height of epithelium.

During 1919–1920 aberrant endometrium, excluding uterine adenomyomata, began to be recognized as a clinical entity and not as just a pathological curiosity. In 1919 Norris[10] reported finding endometrium in the ovary. During the same year Casler[11] reported actual menstruation from uterine endometrium in an ovary. A panhysterectomy and unilateral salpingectomy had been done previously for adenomyomata of the uterus. Drainage was made through the vagina. Subsequent menstruation occurred through the sinus in the vagina caused by the drainage tube. A second operation was done and typical uterine endometrium was found to have developed in the remaining ovary. During 1919–1920 Cullen[12] published several papers on the distribution of adenomyomata containing uterine mucosa. He had found aberrant endometrium in ten places. Mahle and MacCarty[13] reported from the Mayo Clinic 10 cases of adenomyomata arising outside of the uterus. It is interesting to note the distribution of these tumors. They were found in the unbilicus (1), abdominal wall (2), sigmoid colon (1), groin (2), and rectovaginal septum (4). During this period the thousands of cases in which aberrant endometrium occurred in the ovaries evidently escaped unrecognized.

In 1921 Sampson published the first of a large series of very interesting and instructive articles on aberrant endometrium. I believe one is justified in saying that Sampson is chiefly responsible for the recognition of endometriosis as a definite clinical entity. He has also presented an entirely new theory[16] for the origin of aberrant endometrium. According

to his theory some of the mucosa from the lining of the uterus or Fallopian tube becomes detached and travels out through

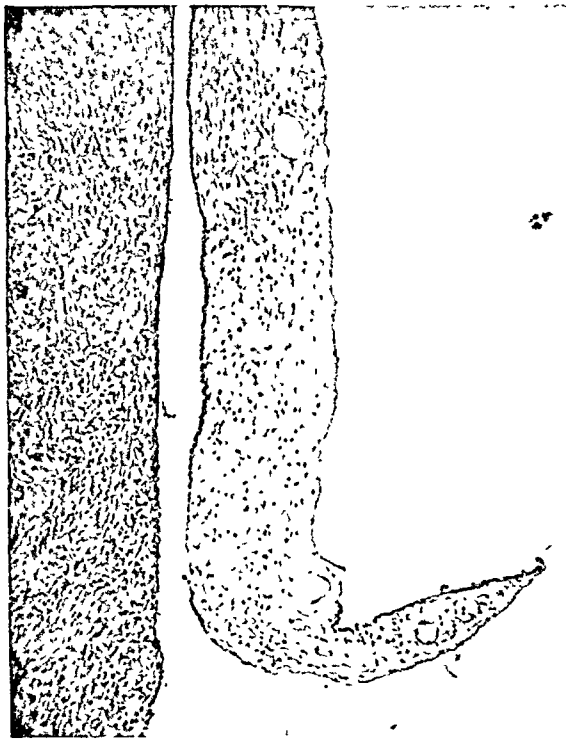


Fig. 3. Another area from same ovary as shown in Figure 1. Invagination of epithelium is again occurring but parallel to surface of ovary instead of perpendicular to it. A variation in height of epithelium can also be seen in this picture.



Fig. 4. Another area of same ovary shown in Figures 1 and 3. Three small "endometrial" cysts are lined principally with high columnar ciliated epithelium. They represent a more advanced stage in metaplasia of germinal epithelium into aberrant endometrium.

the Fallopian tubes to be deposited on the surface of an organ in the peritoneal cavity. Here, he believes, it becomes implanted and grows, infiltrating its host. Before the birth of this theory the aberrant endometrium was believed to have arisen from remnants of either the Wolffian or Müllerian duct, or from a metaplasia of the pelvic peritoneum. The implantation theory of Sampson became very popular from the time of its introduction and has remained so up to the present. Until Novak[15] voiced his objection in 1926 the implantation theory stood more or less generally accepted for the origin of at least some of the aberrant endometrium. Novak believes that the aberrant endometrial tissue results from a transformation of the germinal epithelium or pelvic peritoneum.

INCIDENCE

There are a good many references to aberrant endometrium in the medical journals today, but it is not yet universally recognized nor is the true incidence appreciated. The general surgeon in particular is overlooking this not infrequent condition. So much has been written on the histogenesis of the misplaced endometrium that its incidence has not been sufficiently emphasized. Many excellent men admit that they are finding large numbers of these cases which they have previously overlooked.

Before a discussion of the real incidence of aberrant endometrium it is important to bear in mind that in the great majority of patients the only lesions are small symptomless cysts which are readily overlooked unless an effort is made to find them. They are most frequently situated in the ovary or somewhere on the pelvic peritoneum. These cysts vary in size from

those that can only be seen by the microscope to those about 1 cm. in diameter. Since in so many cases the cysts are first discovered at the routine microscopic examination of the tissue removed, there must be an enormous number that are missed entirely and would only be found by serial sections of the tissue removed. The number of patients in whom the aberrant endometrium is fairly extensive and probably producing symptoms is small as compared to the large number of patients with the small symptomless lesions. For these reasons the incidence reported by different men depends on the interest manifested in this condition and the efforts made to find these lesions. Sampson reports the greatest incidence because he looks for endometriosis in every patient. Sampson found 159 cases of endometriosis in 664 abdominal operations for pelvic disease in three years.

An attempt was made by the author to collect all of the cases of aberrant endometrium from the records of Touro Infirmary from January, 1927 to January, 1931. During this time there were approximately 1100 operations for pelvic disease and aberrant endometrium was found in 135 patients. I do not think that these were all of the patients that had aberrant endometrium because many of the lesions were first found by microscopical examination of the tissue removed at operation. The sections were made at random through this tissue and many areas of aberrant endometrium must have been missed. The condition was undoubtedly not recognized many times at operation when no tissue was removed.

It is interesting to compare the incidence of this condition to the occurrence of other pelvic disease. It was estimated that 35 patients, excluding the ade-



FIG. 5. Section of an ovary showing a metaplasia of germinal epithelium. Epithelium below covering surface of ovary can be seen to be high columnar. Note change to cuboidal and flat and again to a higher type in depth of invagination. Also notice inflammatory exudate on surface of ovary. Some believe that this might be acting as stimulus causing metaplasia.



FIG. 6. Greater magnification of an area shown in Figure 5. Histology of epithelium can be plainly seen in this photomicrograph.

nomyoma group, had aberrant endometrium with adhesions sufficiently extensive to have produced at least part of the



Fig. 7. "Endometrial cyst" on surface of an ovary. This represents a still more advanced step in metaplasia of germinal epithelium of ovary. On right and below, epithelium and stroma is such as to resemble tubal endosalpinx. On left and above, cyst is lined by flat epithelium.



Fig. 8. Area of transition from low to higher epithelium in "endometrial cyst"; greater magnification of area of cyst shown in Figure 7.

patients' symptoms. Some other common pelvic diseases occurring in Touro Infirmary during the time this series was taken are as follows:

Uterine fibroids..	497
Chronic salpingitis.	280
Uterine displacements	202
Cystadenomata of ovary .	40

Thus it can be seen that in this series, while aberrant endometrium is not as frequent as some other common pathological conditions, it is not to be considered a rarity.

HISTOGENESIS

The theories concerning the origin of aberrant endometrium have been numerous. In the earlier literature two were outstanding, viz.: from a displacement of a portion of the Wolffian duct or from a part of Müller's duct. In 1896 von Recklinghausen reviewed the literature and with the report of many new cases came to the conclusion that adenomyomata of the uterus were derivatives of the Wolffian duct. He thought he could recognize a close similarity between the glandular part of the adenomyomata and the elements of the Wolffian duct. In many places the glands were arranged in such a manner as to resemble glomeruli and von Recklinghausen called these areas pseudo-glomeruli.

Russell[6] believed the epithelium he found in the ovary, which was really aberrant endometrium, was due to "an anomalous point of development of portions of the Müllerian duct in the germinal epithelium." Cullen[4] was of the opinion that the adenomyoma he found in the round ligament in 1898 was due to an abnormal embryonic deposit of a portion of Müller's duct.

In 1908 Cullen[2] clearly demonstrated the invasive qualities of endometrium and demonstrated the true origin of into the pelvic cavity. Sampson believes the endometrium becomes implanted where it is deposited and begins growing and



FIG. 9. Area of rather typical uterine type of aberrant endometrium found on Fallopian tube of same patient from which "endometrial cyst" shown in Figure 7 was taken. Glands and stroma in this area closely resemble uterine endometrium while in upper left hand corner musculature of tube can be seen.



FIG. 10. Section through appendix demonstrating aberrant endometrium.

uterine adenomyomata. In 50 uncomplicated cases of adenomyomata of the uterus he was able to demonstrate in every instance a definite connection between the glandular structure in the musculature and the lining of the uterine cavity. This was done by making a large number of sections through each uterus.

At the present time there are two popular theories for the origin of aberrant endometrium within the pelvis, exclusive of the adenomyomata of the uterus. These theories are the implantation and the coelomic. Sampson is the originator of the implantation theory. He believes that bits of the uterine endometrium set free at the time of a normal menstruation or following a curettage make their way through the Fallopian tube and fall free infiltrating. This more often occurs on the ovaries, he believes, due to the proximity of the fimbriated extremities of the tubes to the ovaries. He says that regurgitation through the tubes is often assisted by conditions tending to cause an obstruction to the normal menstrual flow. The incidence of endometriosis has been reported greater in patients with fibroids, pelvic infections and displacements of the uterus, all of which might cause an obstruction to the normal exit of the menstrual blood. Sampson believes that the endometrial transplants menstruate and sometimes form cysts which contain the products of menstruation. Sometimes, he has shown, these cysts rupture and their contents are spread over the peritoneal cavity. The endometrium in these cysts becomes implanted in fresh places on the peritoneal surface and forms daughter cysts. He says: "It seems to me that the condition

found in many of these specimens is analogous to the implantation of ovarian papilloma or cancer on the peritoneal surface of the pelvis from the rupture of an ovarian tumor containing these growths."



Fig. 11. Aberrant endometrium in appendix. Higher magnification of area shown in Figure 10. Endometrial glands and stroma are buried in musculature of appendix.



Fig. 12. Section through isthmic portion of Fallopian tube demonstrating aberrant endometrium on its surface and deep in its wall

Sampson's implantation theory has probably been the most popular of the several theories since its introduction because, I believe, it is the simplest and easiest to understand. I do not think its popularity necessarily implies that it is the true explanation for the aberrant endometrium. Sampson[17] summarizes the facts in support of his beliefs:

1. They are of frequent occurrence (64 instances in 296 abdominal operations for pelvic conditions in one year).

2. They apparently develop during the menstrual life of women and are most frequently found in patients between thirty years of age and the menopause.

3. The histological structure of the endometrial elements in these implants, including the incidence of ciliated epithelium, is identical with that found in the invasion of the uterine wall by the mucosa lining its cavity.

4. The tubes are usually patent and even though occluded the implantation could have occurred before the tubes became closed.

5. The uterus in these patients is often retroflexed, or contains leiomyomata and polyps, conditions which might sometimes favor a backflow of menstrual blood through the tubes.

6. Occasionally blood may be observed escaping from the fimbriated ends of the tubes of patients operated upon during menstruation. The microscopic examination of this blood may show epithelial cells. The histological study of sections of the tubes may also show epithelium and bits of endometrial stroma lying free in the lumen of the tube.

7. As the study of ectopic endometrial tissue in the pelvis demonstrates that implants may sometimes arise from this, it is natural to assume an implantation origin for the former, if a source could be found. Menstrual blood escaping through the tubes indicates a source.

8. They are often found in different stages of development in the same individual, thus

suggesting repeated implantations from the original source or from other implants.

9. The early implants are found on structures in the pelvis which would naturally be reached by blood escaping through the tubes and they are most frequent on the structures in close anatomical relation with the fimbriated ends of the tubes. They may be present only on the peritoneum or only on the ovaries, but are usually found on both. Their distribution is often bilateral, as occurs in pelvic inflammatory lesions of tubal origin.

10. Similar growths have been observed in the scar of the abdominal incision of patients in whom the uterine cavity has been opened, thus affording the opportunity of implanting bits of uterine mucosa in the tissues of the abdominal incision.

11. Jacobson has reproduced lesions similar in many respects by the *autotransplantation* of bits of the uterine mucosa of the rabbit into the tissues of the pelvis.



FIG. 13. Aberrant endometrium in Fallopian tube. Higher magnification of an area shown in Figure 12: Notice endometrium-like glands are surrounded by musculature of tube.



FIG. 14. Aberrant endometrium in tube. Greater magnification of an endometrial gland from an area of Figure 13. Notice lack of any "endometrial" stroma and the cilia on epithelial cells. Glands are resting on musculature of tube.

The chief opponent of this theory is Novak,[15] who makes out, as Maes[19] says, "an excellent case for the opposition." In the histologic study of many hundreds of Fallopian tubes with particular reference to their contents, Novak found only seven which contained particles of uterine mucosa lying free in the lumen. None of the women from whom these seven tubes were removed were menstruating, most of them being many days from this period, and in no instance did the endometrium in the tubes have the appearance of endometrium thrown off at menstruation. In 5 of the 7 cases the particles of the free endometrium were so large that it would seem almost impossible for them ever to have entered the tiny uterine orifice of the tube. Novak believes that the tissue was moving towards the uterus rather than away from it, endometrial tissue being found in the ovary in 2 of these patients. In several other cases only a very small amount of

ovarian tissue was removed and subjected to examination.

Novak does not believe that blood regurgitates through the tubes during menstruation. In 13 patients operated upon by himself during menstruation, he has not seen regurgitation in one. He says that histologic examinations of tubes removed during menstruation characteristically show no blood in their lumen. He calls attention to the fact that no blood has been found in the pelves of thousands of women operated upon by thousands of surgeons immediately after menstruation, although it would hardly have had a chance to be absorbed during this time. He also calls attention to the absence of any frequent peritoneal reaction, as after a ruptured tubal pregnancy, that we would expect to get with the hemorrhage into the cavity from the rupture of a chocolate cyst or spilling of blood regurgitated through the tube.

Novak also questions the retrograde transportation of endometrial tissue into the tube. He thinks the ciliary current to be more important than muscular peristalsis in the passage of the ovum from above downward. He believes antiperistalsis is rare and present only in conditions of overdistention of the tube as proved by recent investigation. He calls attention to the fact that the uterine portion of the tube measures only 0.5 to 1 mm. in diameter so that it would really seem almost incredible that this opening could admit such large pieces of endometrial tissues as are seen in his specimen. He says that if a piece of endometrial tissue made its way through the uterine os of the tube it would still have an almost impassable passage through the very fine lumen of the interstitial portion of the tube; the latter passing in a gently curved or sinuous fashion through the thick musculature of the uterus.

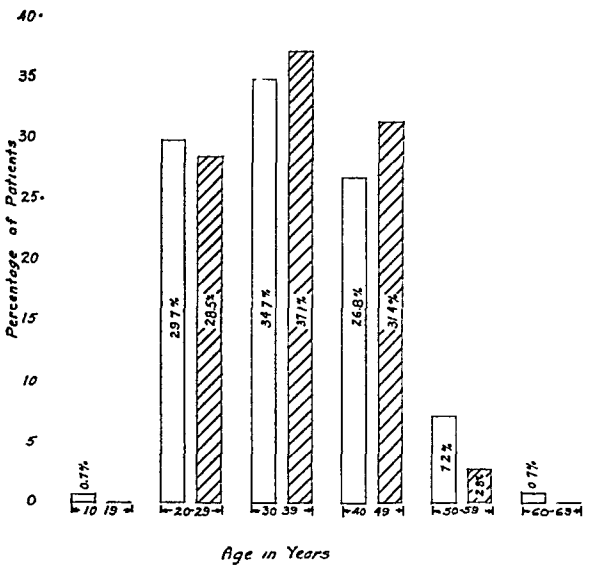


Fig. 15. Age incidence of aberrant endometrium. All cases, plain; severe cases, shaded.

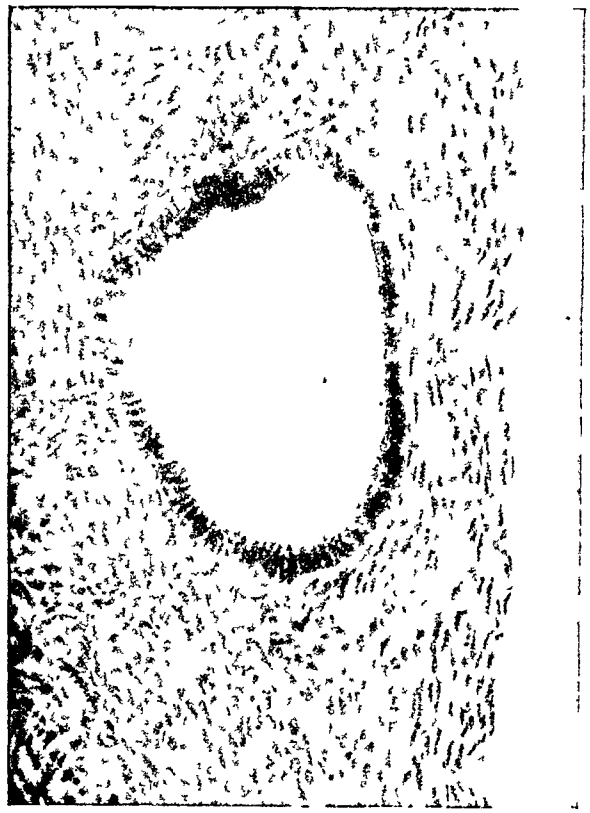


Fig 16 "Endometrial cyst" found on surface of ovary in youngest patient.

In this connection Sampson[20] says:

> I am convinced that the manipulation of the uterus and tubes, incident to pelvic operations, may at times detach uterine and tubal epithelium and cause it to be transferred not only to the abdominal wound, but also to the tissues of the pelvis. This is particularly true

for endometrial tissue if the abdominal operation has been preceded by a curettage. In such instances I have frequently observed blood from the ovary to the uterus it must take longer for tissue to pass in the retrograde direction.



FIG. 17. Endometrial gland and stroma buried deep in musculature of uterus in oldest patient in this series.



FIG. 18. Aberrant endometrium on peritoneal surface of Fallopian tube. This tissue was removed from patient during course of normal pregnancy; Case III.

escaping through the fimbriated ends of the tubes during the abdominal operation. I have also seen instances of peritoneal endometriosis which apparently had resulted from a previous operation.

Blood has been reported at the fimbriated ends of the Fallopian tubes in patients operated upon during the normal menstrual periods by Danforth,[21] Curtis[21] and others.

Blood has been reported at the fimbriated ends of the tubes following curettage by Goodall,[21] Heaney,[21] and others.

Novak questions the ability of the endometrium cast off at menstruation to grow on the ovary or peritoneum. He says that the tissue thrown off is degenerating and doubts that it will have sufficient vitality, after traversing the tube, to grow where it falls. Since it takes several days for the ovum to pass

Novak further objects to the theory because:

1. The endometrium is not infrequently found chiefly and perhaps exclusively, in parts of the pelvis or elsewhere, where implantation from the tube would not be easy;
2. It is often present in extremely small amounts, in spite of the supposed monthly regurgitation and the proliferative tendency of the tissue;
3. It is characteristically confined to the pelvis, even when extensive endometriosis is present, unlike cancer which can implant itself over the entire abdominal cavity.

The coelomic theory has been known in one form or another for a long time. Novak[15] speaks of it as a concept rather than a theory. He describes it very clearly. He says:

It is possible, as some have suggested, that the occurrence of ectopic endometrium at

various points in the pelvis is due to the presence of celomic rests in these regions. But it is just as possible that, aside from the



Fig. 19. Section of Fallopian tube showing villi in patient with an extra-uterine pregnancy.



Fig. 20. "Endometrial cyst" on ovary in same patient from which tissue shown in Figure 19 was taken. No definite "endometrial" stroma about this cyst.

"rest" theory, the normal germinal or other epithelium of the ovary, or the normal pelvic peritoneum, may under the proper combination of endocrine and other conditions, be quickened to various differentiation phases abnormal for the tissue concerned.

This is no mere speculation, for embryologists, on the basis of careful observations, accept the view that there is a definite and intimate relation between all of the epithelial tissues arising from any one of the embryonic layers. Each one of the original embryonic layers possesses developmental potentialities, which, for various organs, are limited at various stages as a result of certain physical or chemical factors, either local or general. It thus is evident that the potency of the cells is not used up, so that if, in later life, the same conditions arise which are responsible for the original development of the cells in question, they may be activated into new growth. For example, a germinal epithelial cell retains a considerable degree of unused developmental potency, and with the proper activation, can develop further into an endometrial or tubal epithelial cell. The same statement may be made of the pelvic peritoneum. What the activating conditions are we cannot say, except that they are almost certainly of endocrine nature. As already stated, these views are not merely speculative, for, as Fischel has emphasized, they have considerable support from embryologic studies.

Applying this conception to the subject matter of this paper, one can readily understand why such an intimate relationship exists between the cells of the pelvic peritoneum, the germinal epithelium, the tubal mucosa, and the endometrium, for they are all descendants of a common mother tissue, the celomic epithelium of the urogenital folds. In this sense the endometrium and endosalpinx are to be looked upon as merely a modified peritoneum, or a modified germinal epithelium, so that it is not so startling to think that either peritoneum or germinal epithelium, plus the necessary but as yet unknown biologic activating stimulus, may give rise to more or less typical Müllerian mucosa . . .

A consideration of possible importance lies in the fact that the activating hormones are

probably found in the ovary itself, and that the frequency of germinal epithelial metaplasia may perhaps be due to its proximity to the source of supply of the hormone—that perhaps there is a greater intensiveness of endocrine action in the ovary itself than in more distant tissues.

Novak thinks he is able to identify many of the stages of differentiation from the germinal epithelium to the uterine endometrium. He has found areas of epithelium growing in the ovary which closely resemble the endometrium of the fetal or infantile uterus. He believes the reason all of the aberrant endometrium does not menstruate is that some of it has not differentiated sufficiently to be excited by the hormone that causes the cycle in the uterine endometrium. Sampson believes that menstruation occurs in the cysts and the blood is reabsorbed. In support of this view he has been able to demonstrate blood pigment in the tissues around the cysts. We know definitely that all aberrant endometrium does not react to pregnancy as does the uterine endometrium, Sampson reported post-salpingectomy endometriosis in 3 patients that were operated upon for tubal pregnancies. In only one of these was there a decidual reaction in the misplaced endometrium. However, in the other 2 patients the aberrant endometrium probably arose from the tubal endosalpinx. In the series reported by the author tissue resembling aberrant endometrium was found associated with pregnancy in 2 patients. In neither was there a decidual reaction in the misplaced endometrium-like tissue. The absence of a decidual reaction in aberrant endometrium may be explained in the same manner that the failure to menstruate is accounted for, i.e. the aberrant endometrium has not sufficiently differentiated to react to the hormone which causes the uterine endometrium to undergo a decidual reaction.

Novak has also been able to demonstrate tissues closely resembling the uterine and tubal endometrium growing next to each other and being continuous with one another. This is additional evidence in support of the metaplasia theory, he believes. He presents an excellent photomicrograph of such an area in one of his articles.[15] In the histological study of aberrant endometrium one is frequently able to find an area of endometrial like tissue the epithelium of which can be seen to grade off into the germinal epithelium covering the ovary. Russell[6] demonstrated such an area in his original report and such a condition is seen in Figure 6.

It seems very likely that both theories are correct and the aberrant endometrium has its origin in both manners.

PATHOLOGY

Aberrant endometrium has been reported in a large number of places. It has been described on all parts of the peritoneum in the pelvis. It has been described frequently on the large and small bowel as intestinal adenomata or adenomyomata. It has been frequently found on the appendix. Aberrant endometrium has not infrequently been reported in the groin, labia majora, vagina, umbilicus, and abdominal wall. Aberrant endometrium has been reported in the sac of an inguinal hernia.[25] With one exception it has always been found below the level of the umbilicus. Hosoi and Meeker[22] reported a case of endometrial carcinoma of the mid-portion of the transverse colon. This portion of the intestine might easily have been much lower in the abdomen when the patient was in the erect position.

The aberrant endometrium sometimes infiltrates the organ on whose surface it is located and is found buried deep in this organ. Thus it not infrequently involves the bowel, especially the sigmoid colon and rectum, to such an extent that it produces an obstruction. It has been found to infiltrate the bladder sufficiently to be recognized at cystoscopy. Hosoi and Meeker[22] reported finding 10 cases of

endometriosis of the bladder wall reported in the literature.

Aberrant endometrium is not infrequently found in scars from abdominal operations, usually following operations on the female genital organs, but several cases have been reported following simple appendectomies. Hosoi and Meeker[22] were able to find 87 cases of endometriosis in laparotomy scars reported in the literature.

Hosoi and Meeker[22] were also able to find epithelial inclusions of endometrial type in lymph glands as reported by several observers.

Adenomyomata of the rectouterine and rectovaginal septum are not infrequently found. Adenomyomata of the uterus are too well known and their incidence too well appreciated to be considered here.

The histological picture of the lesions described as aberrant endometrium varies from single cysts, too small to be seen by the naked eye, lined with cuboidal or columnar ciliated epithelium and surrounded by little or no stroma, to tissue histologically identical with tubal or uterine endometrium. Any gradation from one extreme to the other can be found. As Novak has shown, areas can be found which resemble the fetal or infantile endometrium. Areas can be found where rather typical uterine endometrium adjoins and is continuous with rather typical endosalpinx.

The cases of aberrant endometrium in the series reported by the author were classified as follows:

	Cases	Per Cent
Total number of cases	135	
Adenomyomata of uterus	21	15
Peritoneal endometriosis	114	85
Aberrant endometrium fairly extensive with adhesions sufficient to produce symptoms	35	26
Aberrant endometrium less extensive with few or no adhesions	67	49
Aberrant endometrium clinically not found microscopically	11	8

Most of the lesions in the last group were small superficial cysts on the ovary or pelvic peritoneum which were wiped away at the time of operation. All of these patients had more than one lesion of aberrant endometrium and the diagnosis was only accepted when the patients were being treated by men who had a high percentage of correct diagnoses for the entire series.

The distribution of aberrant endometrium in the series reported by the author was as follows:

	Cases
Ovary	99
Tubes	20
Adenomyomata of uterus	21
Anterior or posterior surface of uterus and broad ligament	8
Rectum and sigmoid	7
Cul-de-sac	5
Appendix	4
Cecum	2
Ruptured chocolate cysts	3

It has been the general observation that aberrant endometrium occurs most frequently on the ovary. Sampson says this is because of its close proximity to the fimbriated extremity of the tube. Novak says that it might be because the hormone which is the cause of the epithelial metaplasia has its home in the ovary itself.

The distribution of the aberrant endometrium reported by King[23] was as follows:

	Cases
Rectovaginal space	52
Petitoneum	26
Ovary	23
Uterus	17
Tubes	4

In reviewing the routine microscopical sections of the tissue removed at operation I was impressed with the frequency with which a diagnosis was made first by the pathologist. It was evident that many of the lesions were not being recognized by the surgeons at the time of operation. An examination of the records from this angle revealed this interesting information:

	Surgical Services	Gyneco-logical Services
Total cases	43	89
Cases recognized at operation	1	52
Percentage of diagnosis	2	58

That the general surgeon is overlooking aberrant endometrium is well shown in this series for only one case out of almost 50 was recognized at the time of operation.

To recapitulate, it is impossible to recognize every case grossly or at operation because the endometrium is sometimes so small that it can only be seen by the microscope. This is illustrated by the series of Dr. C. Jeff Miller who was kind enough to let me review his cases. Out of 57 cases the aberrant endometrium was so small as to be found by the pathologist in 11 instances. During these four years Dr. Miller had recognized 46 cases. So we might say in 19 per cent of this series the lesions are so small that they cannot be recognized grossly. I do not think Dr. Miller has overlooked any lesions large enough to be seen readily because he has been particularly interested in this subject for several years and has been watching for this form of pathology.

The relation of other pelvic diseases to aberrant endometrium has been frequently emphasized. Several authors thought they were able definitely to correlate a relationship between aberrant endometrium and uterine fibroids, malpositions of the uterus or chronic pelvic infections. It was and is believed that these diseases in the pelvis are in part responsible for the aberrant endometrium by obstructing the normal exit of the menstrual flow and causing the blood and desquamated epithelium to back up into the Fallopian tubes and into the peritoneal cavity. On the other hand there are some who believe that the inflammation, *per se*, causes an endometrial metaplasia. Associated pelvic conditions were found in this series as follows:

	Cases	Per Cent
Myoma uteri	62	46
Uncomplicated with exceptions of adhesions in pelvis	43	
Pelvic inflammatory diseases	19	
Including pus tubes	2	
Displacements of the uterus	12	8
Pelvic inflammatory diseases not associated with myoma uteri	32	
Total pelvic inflammatory diseases	51	37
Only adhesions in the pelvis besides aberrant endometrium	15	11
Other cysts in the ovary	54	

Associated pelvic conditions reported by other men are as follows:

	Keene-Kimbrough Per Cent	King Per Cent	Smith Per Cent
Myoma-uteri	55.4	22	41.6
Chronic salpingitis	20	39.7*	
Adherent retroverted uterus	14.5	21	25.8

* Evidence of some inflammation in the pelvis.

SYMPTOMATOLOGY

After reviewing the case histories of the patients in this series the author has come to the conclusion that there are no constant characteristic signs or symptoms of aberrant endometrium. Most of the symptoms given by patients coming to operation are produced by some associated pathological condition. The symptomatology may be classified as follows:

1. *Age Incidence.* Aberrant endometrium is believed to develop during the menstrual life of women and Sampson says endometriosis is most frequently found in patients between thirty years of age and the menopause. In the series reported by the author the youngest patient was sixteen years old. The lesion in this patient, however, cannot positively be called aberrant endometrium. The diagnosis was made on the basis of a single cyst lined with cuboidal ciliated

epithelium and not surrounded by any characteristic stroma. A photomicrograph of this lesion is shown in Figure 15. The oldest patient was sixty-one years old and had a rather diffuse adenomyoma of the uterus. King[23] reported finding an adenomyoma in the uterus of a patient sixty-four years old. The respective ages of the patients in this series are shown in Figure 15. The age incidence by decades reported by Keene and Kimbrough[26] is as follows: third decade, 18.6 per cent; fourth, 49.1 per cent; fifth, 25.4 per cent; and sixth, 6.8 per cent.

2. *Sterility.* The importance of sterility as a symptom in these patients has been emphasized. Keene and Kimbrough[26] reported 40.9 per cent of their patients were sterile; Smith 20.6 per cent; and Shirer 84.2 per cent. In the present series 84 per cent were married; 30 per cent of these were sterile. One patient bore twelve children. It must be remembered however, that in many of these patients the aberrant endometrium was considered to be too small to produce symptoms. If only the patients with rather marked endometriosis were considered the incidence of sterility was practically the same, 29 per cent. It would seem then that the associated pathology, fibroids, pelvic infections, uterine displacements, etc., must play a part in the high incidence of sterility of these patients. Twenty per cent of the patients with marked endometriosis were unmarried.

3. *Irregularities of Menstruation.* The consensus of opinion among writers on the subject is that aberrant endometrium, *per se*, produces no characteristic menstrual irregularity. In the series reported by the author the menstrual disorders complained of by the patients were as follows:

	Cases	Per Cent
Menorrhagia	30	22
Metrorrhagia	15	11
Dysmenorrhea	35	26
Amenorrhea after menopause	4	3
No menstrual disturbance	69	51

If only the patients having rather extensive degrees of aberrant endometrium were considered the menstrual irregularities were as follows:

	Cases	Per Cent
Total	35	
Menorrhagia	11	31
Metrorrhagia	3	8
Dysmenorrhea	11	31
No menstrual disturbance	17	48

I do not believe there is enough difference in the percentages of these groups to say that the more extensive endometriosis could produce certain menstrual irregularities. In a series of endometriomata reported by King the menorrhagia could be explained in 75 per cent of the patients by some associated uterine lesion. Keene and Kimbrough conclude that profuse menstruation is usually a symptom of the complicating lesion rather than of the endometrioma itself.

4. *Dyspareunia.* It is impossible to say what percentage of patients in this series presented this symptom, because they were not questioned routinely on this point. There were several patients, however, who gave this symptom as their chief complaint.

5. *Other Important Symptoms.* In addition to the symptoms already mentioned there were two which were rather prominent among the patients' complaints. These symptoms with their relative incidences were as follows:

	All Cases		Extensive Endometriosis	
	Cases	Per Cent	Cases	Per Cent
Pain and tenderness in lower abdomen not associated with periods	38	28	11	31
Leucorrhea	33	24	6	17

Probably the most important individual symptom, when it does occur, is pain

and tenderness over the site of the growths during the menstrual period. This is particularly significant if it can be definitely differentiated from uterine cramps. For instance if the pain and tenderness occurs during menstruation in the scar of a former laparotomy incision or in a nodule in the groin or labia it is very significant. Pain and tenderness do not occur, however, at the menstrual period in all cases of aberrant endometrium. The conclusion I have drawn from the review of the case histories in this series and from the stories of the comparatively few patients I have seen myself is that pain and tenderness at the site of the aberrant endometrium is rather uncommon. Novak has shown, as was said before, that some of the aberrant endometrium resembles the fetal or infantile types and only that which has become sufficiently differentiated reacts to the hormone producing menstruation. Certainly it would not seem probable that the tissues resembling the tubal endosalpinx would menstruate as would the uterine endometrium. In the majority of cases the tissues removed during the menstrual and premenstrual periods have no similarity to the stage of the menstrual cycle found in the uterine endometrium as can be seen from the uterine scrapings. In fact I have not been able to find aberrant endometrium having the appearance of the premenstrual and menstruating uterine endometrium. I do not mean to imply that all aberrant endometrium does not menstruate. I only mean to emphasize that menstruation would seem to be the exception and not the rule. Therefore the symptoms of pain and tenderness over the site of aberrant endometrium, arising from overdistention of the cysts, is not common, but when it does occur it is very significant.

REPORT OF INDIVIDUAL CASES

In this series there are several patients warranting especial attention.

Case I. The youngest patient was sixteen years old. Miss S. B., a white female, was admitted to the hospital with a diagnosis of chronic appendicitis. Her menstrual history was normal. An appendectomy was done and on examination of the pelvis one ovary was found to contain many small cysts. This ovary was resected and the microscopic examination of the tissue removed demonstrated the presence of aberrant endometrium. (See Fig. 16.)

Case II. The oldest patient in this group was sixty-one years old. Mrs. K. J., a white female, had passed the menopause several years previously. Shortly before admission she began to pass clots of blood. Examination revealed multiple fibroids. A supravaginal hysterectomy was done. The uterine endometrium was atrophic and endometrium was found throughout the wall of the uterus. A diagnosis of adenomyoma was made. (See Fig. 17.)

Case III. One patient was operated upon during a normal pregnancy. Mrs. J. S., a white female, forty-two years old, was married. She had two normal children. Twelve years previously she was operated upon at which time her uterus was suspended, the right ovary resected and the appendix removed. Six weeks before admission she received a blow in the abdomen and shortly afterwards began to have irregular bleeding. Examination suggested fibroids, a panhysterectomy was done and a normal pregnancy was found as well as aberrant endometrium on the ovaries. (See Fig. 18.)

Case IV. Endometriosis was discovered in one patient operated upon for an ectopic pregnancy. Mrs. H. B., a white married female, twenty-five years old, believing herself pregnant attempted an instrumental abortion. She was admitted to the hospital two days following the attempt. At this time she had no discharge nor was she suffering pain. On vaginal examination an extra-uterine pregnancy was suspected. After dilatation and curettage the abdomen was opened. The uterus was found bound down in the right side of the pelvis, the right tube enlarged and the middle third ballooned out with a hard nodular mass. The ovary was cystic. A right salpingo-oophorectomy was done. Microscopic studies revealed products of pregnancy in the uterine scrapings; in the ovaries a developing corpus

luteum, follicular cysts and aberrant endometrium; and in the tube an extra-uterine pregnancy. In neither this nor the preceding patient was there any evidence of a decidual reaction in the misplaced endometrium.

Sampson reported 3 cases with post-salpingectomy endometriosis that were operated upon for tubal pregnancies. In two of these there was no evidence of a decidual reaction in the misplaced tubal mucosa of the uterine cornua; nor was it present in the uterine mucosa in either case. In the third patient the endometrium in both cornua showed a marked decidual reaction as did also the mucosa of the uterine cavity.

There were 3 patients in the present series who had ruptured chocolate cysts at the time of operation:

Case v. Miss I. C., a white female, twenty-three years old, was admitted to the hospital with a chief complaint of acute pain in the right lower quadrant of the abdomen. During the day preceding her admission she had suffered from nausea and vomiting, although she had been up and about. At about midnight the pain began in the right lower quadrant of the abdomen and radiated to the left side. Menstruation was normal. There was some tenderness and rigidity over the entire lower abdomen. A vaginal examination was not made, a rectal examination was unsatisfactory. The abdomen was opened with a right rectus incision and the intestines and pelvis were found covered with a chocolate colored non-clotted material which was found to have come from a ruptured chocolate cyst of the left ovary. About one-half of the contents of the sac had been emptied and there was also some bleeding from an implant in the cul-de-sac. There were one or two small superficial implants on the right ovary which were readily wiped off.

Case vi. Miss A. C., a white female, twenty-one years old, was admitted to the hospital with a chief complaint of pain in the right lower quadrant of the abdomen. For several months she had suffered from some mild pain in her lower abdomen. The day before her admission she had a sudden attack of severe pain in the lower abdomen which localized to the right side. Nausea and vomiting followed. Menstruation was entirely normal except for occasional cramps. On examination tenderness and rigidity were present over the lower abdomen. A mass could be identified in the left side of the pelvis. The blood count was 20,000 white corpuscles with 91 per cent polymorphonuclear leucocytes. On opening the abdomen a large amount of thick chocolate-colored fluid escaped. A large ruptured chocolate cyst of the left ovary was found densely adherent to the sigmoid and apparently almost ruptured into the bowel.

Case vii. Miss P. R., a white female, thirty-six years old, was admitted to the hospital with a chief complaint of pain in the upper abdomen. The onset of the pain was three days prior to her admission and the pain had progressively grown worse. It radiated from the upper right abdomen to the lower right quadrant. The patient was nauseated but did not vomit. There was no history of any menstrual disturbance or pelvic discomfort. Her temperature was 100°F., pulse 110 per minute. The blood count was 23,000 white corpuscles with 85 per cent polymorphonuclear leucocytes. Tenderness and rigidity were present over both the regions of the gall bladder and the appendix. On opening the abdomen a prune-juice colored fluid escaped. The gall bladder was distended, its walls thickened and there were a few adhesions but there had been no perforations. The appendix was inflamed from the middle to the tip. Exploration of the pelvis disclosed two small subperitoneal fibroids and a large white flabby mass on the left side, which proved to be an ovarian cyst which had ruptured and discharged its contents into the peritoneal cavity. Probably a quart and a half of this unclotted blood was removed. The collapsed sac which proved to be the left ovary was removed. The patient's condition became steadily worse; she developed an intense jaundice, generalized peritonitis and died three days after her admission.

The case histories of the last three patients are interesting from the standpoint of making a diagnosis. In the first and the last the diagnosis should not have been suspected. In the second preoperative diagnosis of aberrant endometrium would hardly have been justifiable. The pathology found in these last three

patients would tend to support Sampson's implantation theory for the origin of the aberrant endometrium. It is not hard to believe that if the endometrium is sufficiently active to grow and form fairly large cysts, it could become implanted on other parts of the peritoneum in the pelvis after rupture of one of these cysts.

DIAGNOSIS

In the majority of cases it is impossible to make a correct diagnosis of aberrant endometrium in the pelvis before operation. Aberrant endometrium in the pelvis sufficiently extensive to warrant radical treatment would cause enough pathology by the production of adhesions to warrant an exploratory operation. It is very essential that the condition be recognized at the time of operation and the necessary treatment instituted. The occurrence of numerous cysts of varying sizes containing a chocolate-colored fluid and surrounded by many adhesions is characteristic. The small solitary cysts about 1 cm. in diameter containing a dark brown or bluish fluid occurring on the surface of the ovary or any organ or organs in the pelvis are diagnostic.

When the lesions are in the rectovaginal septum the diagnosis is sometimes not so difficult. If several dark blue cysts can be seen beneath the vaginal mucous membrane the condition should be assumed to be aberrant endometrium. The patient might or might not give a history of pain or discomfort associated with the menstrual periods.

If the aberrant endometrium occurs in the round ligament outside of the peritoneal cavity it may be felt as a small mass in the inguinal canal or labia majora. This also may or may not react to menstruation.

Aberrant endometrium in an old laparotomy scar is identified mainly by a history of the scar getting painful and tender during each menstrual period. The patient might report that the scar seems to swell during the periods. I have recently heard of a patient who had a little bloody discharge from a sinus in her scar during each menstruation.

TREATMENT

The treatment of aberrant endometrium should depend on the amount of pathology found. The small superficial cysts should be wiped away with a sponge or burned off with a cautery. If the lesion is a little more extensive it should be excised if possible. If the aberrant endometrium is too extensive to be excised completely, retrogression can be assured by removing the ovaries. This should be done, however, only when the patient's symptoms are rather severe and are apparently due to the aberrant endometrium. Sacrificing *both ovaries may give a train of symptoms* which would be worse than the symptoms produced by the aberrant endometrium. In view of the large number of very small symptomless lesions as compared to the relatively few larger lesions producing symptoms, it must be assumed that in many instances the pathological condition remains limited for a long while and often never obtains sufficient size to cause any trouble. In view of this fact it would seem criminal to do a bilateral oophorectomy just because some aberrant endometrium was found. Most of the adenomyomata occurring in old laparotomy scars, around the umbilicus and the labia majora or groin can be excised completely.

SUMMARY

1. From the records of Touro Infirmary 135 patients with some form of aberrant endometrium were found in a series of 1200 patients who were operated upon for some pelvic pathology between January, 1927 and January, 1931.

2. Of these 135 cases 15 per cent were adenomyomata of the uterus and 85 per cent were peritoneal endometriomata. The aberrant endometrium in the pelvis was considered to be extensive enough to produce some of the symptoms in 26 per cent of the patients of the entire series.

3. During the time that this series was taken, there were 497 patients with uterine fibroids operated upon; 280 with chronic salpingitis; 202 with uterine displacements;

and 40 with cystadenomata of the ovary.

4. The belief that the condition was not being recognized by the general surgeon was justified. The correct diagnosis was made at the time of operation in 58 per cent of patients on the gynecological services and in only 2 per cent on the general surgical services.

5. It was estimated that in 19 per cent the lesions were so small that they could only be recognized by the microscope.

6. Fibroids were associated with the aberrant endometrial tissue in 46 per cent of the patients. Displacements of the uterus were mentioned in 8 per cent of the cases. Pelvic inflammatory disease was present in 37 per cent.

7. The youngest patient in the series was sixteen years old, the oldest sixty-one. The majority of patients were between twenty and forty-five years of age.

8. Thirty per cent of the patients were sterile. One patient was the mother of twelve children. Twenty per cent of the patients with marked endometriosis were unmarried.

9. Menorrhagia was a symptom in 22 per cent, metrorrhagia in 11 per cent, dysmenorrhea 26 per cent and no menstrual disturbance in 51 per cent. Three per cent were past the menopause. In the group with extensive endometriosis, menorrhagia was a symptom in 31 per cent, metrorrhagia in 8 per cent, dysmenorrhea in 31 per cent and no menstrual disturbance in 48 per cent. The aberrant endometrium was believed to have caused little if any of the menstrual disorders. The symptoms were believed to have been caused principally by the associated pathology.

10. Dyspareunia was present in several.

11. In one instance aberrant endometrium was found in a normal pregnant woman; in one instance it was found in a patient with an extra-uterine pregnancy. In neither of these patients was there a decidual reaction in the aberrant endometrium.

12. Three patients were operated upon and ruptured chocolate cysts found. In none of the three was this suspected before operation.

13. There are no constant characteristic signs or symptoms of aberrant endometrium. In most instances the associated pathology can account for the symptoms and signs. The most important individual symptom is pain and tenderness over the site of the growths during the menstrual period. This, however, is the exception and not the rule.

14. Aberrant endometrium probably has its origin both from the implantation of uterine mucosa regurgitated through the tube and from a metaplasia of the germinal epithelium or pelvic peritoneum.

CONCLUSIONS

Aberrant endometrium is a not infrequent condition which is still escaping general recognition. It sometimes produces marked symptoms of a varied nature depending on the extensiveness of its growth and the organs it affects.

I wish to acknowledge the assistance of Miss Alba Richardson and members of the record room staff of Touro Infirmary in the compilation of the statistics for this paper.

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[For Remainder of References see p. 336.]

ACUTE INTESTINAL OBSTRUCTION DUE TO FETAL PERITONITIS*

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A MALE infant weighing 7 pounds was delivered by version and extraction. The mother was apparently healthy in every way and in particular there was nothing in her history or examination to indicate tuberculosis or lues and at a later date a blood Wassermann test was negative. The baby's condition after delivery and until the tenth day was apparently normal except for a loss in weight a little greater than the average and a temperature that was ½ to 1 degree subnormal most of the time. Breast feeding was used alone until the eighth day when a supplemental feeding was begun. The bowels moved one to five times a day and there was no regurgitation or vomiting during the first ten days. On the tenth day the abdomen was slightly distended, the temperature was normal. On the eleventh day the baby appeared apathetic, restless, and nursed poorly; the abdomen was more distended and he vomited once; the temperature arose to 101°F. On the twelfth day the abdomen was more distended and vomiting occurred several times. The child was more restless and the temperature arose to 102°F. The diagnosis of peritonitis of unknown origin was made and operation advised.

Laparotomy under light ether anesthesia revealed much free purulent fluid in the peritoneal cavity and a small amount of free feces. A loop of ileum was densely adherent to the right lobe of the liver and in the right flank anterior to the ascending colon. These adhesions were fibrous and very evidently of long standing. The adherent loop and its mesentery were twisted several times at the base, forming a volvulus, and parts of the loop were gangrenous with a resultant perforation. Other dense adhesions were found in the right upper quadrant. The appendix was visualized but was normal except for some reddening. Twelve inches of gangrenous ileum were freed by rather difficult dissection, brought out on the abdomen, excised and a tube sewed in each limb as the child was in too poor condition for a more lengthy surgical procedure. The abdomen was thoroughly drained and loosely closed. The child lived for thirty-eight hours. Vomiting occurred repeatedly, the bowels however moving about ten times before death. Postmortem examination revealed no other congenital anomaly of the abdominal viscera.

About eighteen months later Dr. H. B. Tebbetts delivered the mother of another apparently healthy baby. The subsequent history and findings, the operation and postmortem findings practically duplicated those of the case presented.

A number of causes of acute intestinal obstruction in the newborn have been described. One of the less common but of unique interest is a fetal peritonitis. Neff states: "In fetal life a rare chronic form of peritonitis may occur and is sometimes ascribed to the absorption of toxins or infections from the mother." Lues and tuberculosis have been generally considered the most common primary causes. According to Grulle: "It is the general consensus of opinion that, when present, fetal peritonitis is the result rather than the cause of obstruction" and "in practically all instances the cause may be found in some developmental error."

In the case reported it is believed that a fetal peritonitis had occurred and was the primary factor in the formation of the intestinal obstruction and that the adhesions formed therefrom prepared the way for the volvulus. The reasons are: 1. The very evident chronicity of the adhesions, these being too dense and fibrous to have formed since birth. 2. No apparent atresia or defect in the continuity of the bowel lumen until the volvulus occurred. 3. No embryologic anomaly that can adequately explain the conditions found. The sequence of events probably was this: a peritonitis, developing during fetal life, with adhesions forming between the ileum and other structures; the formation of a volvulus with the fixed portion of the ileum as a pivot; intestinal gangrene; perforation; acute peritonitis and death.

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COMMON ERRORS IN THE DIAGNOSIS OF RECTAL TUMORS*

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TOO frequently we find malignant tumors in the rectum and sigmoid which are inoperable, but which might have responded to treatment if discovered earlier. There are several factors in the examination of the rectum and sigmoid, which, being neglected or misinterpreted, may lead to error in diagnosis, especially of these conditions.

Such remarks as "the *x*-ray examination was negative"; "proctoscopic to 8 inches was negative"; "mass felt or seen at a level of 5 inches"; "there is no pain or symptoms of obstruction"; or "the stool is not ribbon shaped"; or "the bowels move regularly"; are often heard.

A negative finding by fluoroscopic or plate examination of a barium enema is of no value, and this fact is emphasized by the roentgenologist. This can be explained by the anatomical structure of the rectum and sigmoid, which allows a wide variation in size and shape when filled with the opaque enema. There is no typical picture of a normal bowel. Although a cancer mass may almost completely obstruct the lumen of the bowel in the rectosigmoid region, the opaque material passes through rapidly, filling the loops posteriorly, masking the filling defect from view. The irregularities of the mass are filled with the material which also obscures the true condition.

An opaque enema which shows a defect is not sufficient to establish the true diagnosis, as to position and extent of the lesion. Due to the looping and mobility of the sigmoid, the lesion may appear in the plate to be above the reach of the proctoscope and still be visualized at 5 or 6 inches. When an *x*-ray examination is considered necessary, and a defect found, the plate of an ingested opaque meal should be taken in order that the length of bowel above the lesion may be determined.

Incorrect proctoscopic examinations may be attributed to improper position, poor lighting, and unsuitable instruments. The distance the scope is inserted is often misjudged, probably because the examiner is not careful in measuring from the anus itself and may include a portion of the buttocks in the estimate. The same factors may account for the impression of a mass being felt at about 5 inches. Change in the bowel habit may be gradual. If the tumor is above the rectum proper, the fecal stream may dribble through and fill the ampulla, and the patient may still have fairly normal stools. If the mass is above the anorectal line, pain is usually a late symptom and is caused by the tumor encroaching upon surrounding tissues. Bleeding may go unnoticed for many months, or may be attributed to piles, and in the absence of pain does not impress the patient sufficiently, and too frequently the doctor makes the mistake of treating for piles without a proctoscopic examination. Bleeding frequently comes from polyps situated above the reach of the finger or anoscope. Single or multiple polyps, and even simple adenomato, often are precancerous, or have definite malignant changes without giving important symptoms. The finding of parasites in the stools of patients with bleeding and frequency has many times allowed a cancer to grow into an inoperable stage. Amebae have been found in the ulcers of a carcinoma. The shape of the stool means nothing, and depends upon the tone and condition of the opening through which it last passed.

* Read before the Los Angeles Surgical Society, November 20, 1931.

BENIGN NEOPLASMS OF THE STOMACH*

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BENIGN tumors of the stomach are of unusual interest to the surgeon because of their rarity and the serious complications which they may produce as well as the possibility of overlooking them in the course of a routine examination and because they can be dealt with only by surgical means. The symptoms suggested by such tumors are obscure chronic anemia, unexplained indigestion and sudden hemorrhage from the stomach.

It is interesting to note that the diagnosis of benign tumors of the stomach has rarely, if ever, been made before operation. They have usually been discovered as an accidental finding or at autopsy. Some idea of the rarity of the disease may be obtained by the fact that Alfred Tilger (quoted by Myer) in 3500 necropsies reports but 14 benign tumors of the stomach. In the Obruchow Krankenhaus in 7500 necropsies, 4 cases of polyps of the stomach were found. In the Russian hospitals, Tilgar found the percentage to vary from 0.007 to 0.04 per cent. Vegele, in 1908, reported one case of polyadenoma, and in a search of recent German and English literature was able to find a treatment of the subject in the works of Collier, Hanser, Post, and Galland. In French literature Menetrier, Brussard, and Norman have reported cases.

Up to March, 1927 at the Mayo Clinic "there had been 35 cases of benign tumors of the stomach in the males and 23 in the females. The youngest of these patients was eight years of age and the oldest sixty-nine. As to location, 69 per cent were in the pylorus, 26 in the body of the stomach and 5 per cent in the cardia."

These tumors may develop in any part of the stomach and may form on either the inside or the outside of the wall, varying in size from that of a pea to a baby's head. They may arise from any of the three coats of the stomach and may be either sessile or pedunculated, at times simulating carcinoma or sarcoma so closely that they cannot be differentiated except through most careful pathological examination. Some of them even metastasize with characteristics that simulate malignancy.

PATHOLOGY

There are a number of different benign tumors that may occur in the stomach. Among the more important of these may be mentioned mucous polyps, myomata, fibromata, lipomata, lymphadenomata, myxomata, osteomata, fibromyomata, adenomata, cysts of different types and syphilitic tumors.

Mucous Polyps. Microscopically, these are composed of the same tissues as a normal mucosa. According to Fenwick, they are really small adenomata that have undergone cystic degeneration. Two types are distinguished by Menetrier: a superficial one involving the excretory ducts and associated with much lobulation and many cysts from the connective-tissue obstruction of the duct; and a deeper form involving the glandular portion mainly, and showing little or no lobulation and few or no cysts. A mixed type occurs also. Polyps may be single or multiple; the latter may number as many as three hundred. To the multiple form the designation of the polyadenomata or polyposis has been given. Single mucous polyps are found frequently near the pylorus and the multiple in the median portion of the stomach. They form soft, slimy, globular or more or less lobulated, or even cylindrical or mushroom-shaped tumors attached to the walls of the organ by a thin stalk or pedicle, and varying

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in size from that of a wheat grain to several inches in length. The solitary polyp usually measures from ½ to 4 inches; the multiple

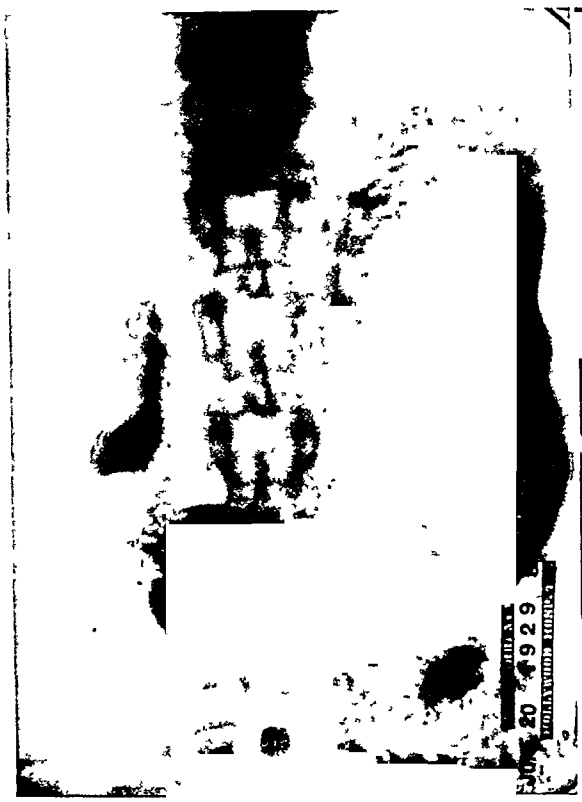


Fig. 1.



Fig. 2.

seldom exceed ¾ inch in length, are fairly uniform in size, and have been compared to a bunch of grapes. The color varies with the vascularity, from a gray to a deep red brown. The surface is smooth and never adherent.

Reports vary as to the frequency of their occurrence. Thus Ebstein found them present 14 times in 600 autopsies, whereas in 7500 autopsies at the Obruchow Krankenhaus only 4 cases of polyps were discovered and Fenwick states they did not exceed 0.2 per cent of the London cases.

As regards the etiology of the gastric polyadenomata, a number of explanations have been advanced but none has been universally accepted. The causes assigned include chronic alcoholism, chronic gastritis, atheroma of the gastric vessels, and other conditions leading to chronic nutritional disturbances of the mucosa. Plausible as these are in individual cases, no one of them would appear applicable to all. The most probable conducive factor is chronic gastritis, which so frequently results in hypertrophic thickening and lobulated swellings of the mucous membrane and finally may lead to the development of smaller or larger polypoid formations. However, Aschoff believes this sequence to be exceptional and that the catarrh is secondary to the polyposis. In this connection, attention may be directed to the association of catarrhal enteritis with polyposis intestinalis and the reported disappearance of the latter after the amelioration of the catarrhal process from colonic irrigation through a colostomy opening.

Myomata. The myoma is a common form of benign tumor of the stomach. Most of these cases, however, proved to be malignant, showing areas of fibro-sarcomatous or myosarcomatous degeneration. Myomata may be single or multiple and are usually situated in the pyloric region,

forming firm rounded tumors. Von Erlach removed a myoma said to have weighed 5400 gm., von Eiselberg one the size of a



Fig. 3

man's head, and Kunze a lipomyoma of 251 gm. The submucous variety may undergo cystic, and the subserous, carcinomatous degeneration. Microscopically they consist of unstriated muscle fibers mixed with strands of fibrous tissue.

Fibromata. These may be single or multiple, and are usually situated in the pyloric region. As a rule they are elongated or club shaped and measure from 1 to 4 inches in length. Occasionally they assume a globular form. Microscopically they either present a papillomatous structure or consist entirely of fibrous tissue covered with thin mucous membrane. Fenwick doubts the identity of reported cases of simple intramural fibromata of the stomach. He believes that they are either ordinary scirrhous carcinomata, fibrosarcomata or keloids formed from the scar of a former ulcer. He says: "We have not been able to find a single case in all the literature of a large fibroid tumor of the gastric walls above suspicion."

Lipomata. The lipomata are very rare, are usually solitary, and are generally situated in the central part of the stomach, on the anterior wall. They originate in either the submucous or subserous coats and, pushing these back, project as small or even very large pendulous masses into the cavity of the viscus or into the abdomen. Very rarely they may be diffused and spread over a large area of the stomach wall. In rare instances they may undergo cystic or malignant degeneration.

Lymphadenomata. This is a peculiar form of gastric tumor due to an overgrowth of the normal lymphoid tissues. It may develop either in the mucosa, forming numerous polypoid and sessile tumors which project into the lumen and infiltrate the walls of the organ, or it may originate externally in the mesenteric glands and invade the stomach through the serosa. The growth may be widely disseminated and frequently there is a general enlargement of the systemic lymphatic glands similar to that occurring in Hodgkin's disease. The pyloric region appears to be singularly free from these growths. They are soft, smooth and creamy white, frequently undergoing superficial ulceration or softening at their base, and even gangrene. Microscopically, they consist of thickened folds of mucous membrane infiltrated with lymphoid elements. The submucous coat is often unaffected.

Myxomata are gelatinous, semi-transparent tumors, encapsulated in the walls of the stomach and covered by mucous membrane. Only 3 cases have been reported, including one by Hansemann.

Osteomata are really fibromata and sarcomata which have undergone partial ossification. The best known case is that reported by Webster in 1827.

Fibromyomata when pedunculated take the form of firm rounded tumors which

vary from the size of a pea to that of an orange, and are attached to the wall of the stomach by a thin pedicle. They may be single or multiple, and are usually situated in the pyloric region.

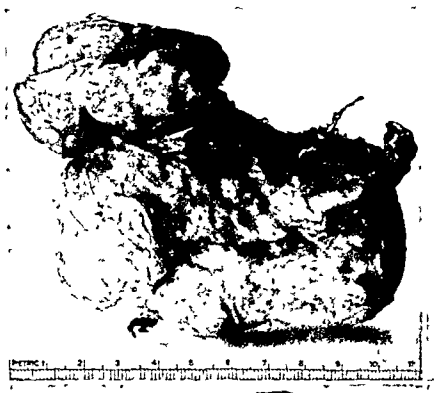


Fig. 4.

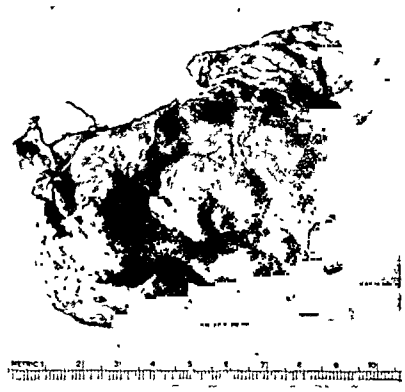


Fig. 5.

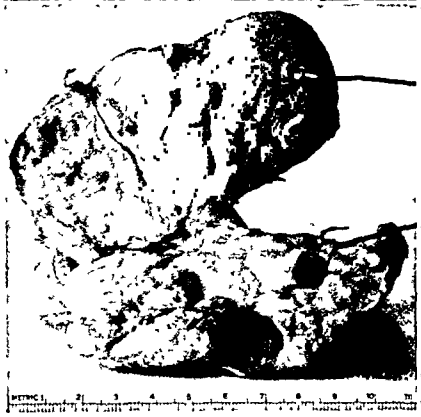


Fig. 6.

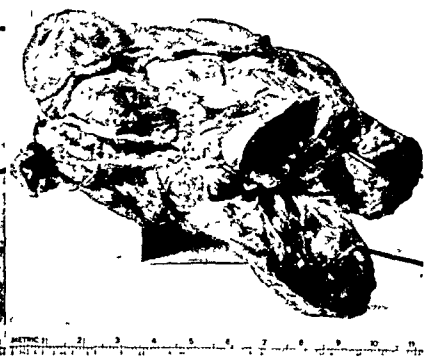


Fig. 7.

Adenomata. These are composed of the same structural elements as are mucous polyps. But while the latter are mainly composed of mucous membrane true adenomata consist of more or less tortuous, irregularly dilated tubular glands supported by connective tissue and well supplied with blood vessels. The overlying mucous membrane is affected by chronic interstitial inflammation. The pedundulated variety is most frequently situated in the pyloric region.

Adenomata form rather firm, smooth, round or somewhat lobulated tumors occasionally cystic in part, the cyst being filled with brown mucous. They may be single or multiple. When multiple, they rarely attain a size larger than a walnut. Single ones vary greatly in dimensions, the largest recorded being a pedunculated one successfully removed by Chaput, which was the size of a fetal head at term.

A most important fact in connection with adenomata is their close association with carcinomata. Not only is it at times very difficult to differentiate between the two histologically, but even where the microscopical evidence favors benignancy, the clinical course and gross appearance may bespeak a malignant type of disease.

Cysts. Seven varieties of cysts have been noted. The most common are retention cysts met in obstruction of the mouth of the ducts of the gastric glands in chronic gastritis. Other varieties that have been described are dermoid, serous, hydatid, blood, lymphangiomatous or chylous cysts, and those cysts formed through degeneration.

Syphilis. According to Fenwick, syphilis may affect the stomach in three ways: (1) by the formation of gummata; (2) by the production of endarteritis, and (3) by exciting chronic inflammation in its mucous membrane. He states that gummata of

sufficient size to attract attention are rarely encountered, and that only a few genuine cases are recorded in the literature on the subject.

SYMPTOMATOLOGY

Benign tumors of the stomach may exist throughout life without evidence of any clinical symptoms. This is particularly the case with polyadenomata. Quite a number of such cases have been reported, the majority, however, having been found at the time of autopsy following death from other conditions. It is a curious and interesting fact that death has resulted in most of these cases from cardiovascular or renal conditions, and that associated with these tumors is often found a condition of chronic hypertrophic gastritis. A number of observers have considered that the disturbed nutrition of the gastric mucosa is an etiological factor in the production of these growths. This, however, has not been proved, for cases have been found in which gastritis was not present. It has also been suggested that the presence of cardiovascular conditions might be a causative factor in the growth of these tumors, but this is not probable. When these benign tumors are situated near the pylorus they may be provocative of most serious symptoms. Various degrees of stenosis may be produced. Death may result from hemorrhage or by ulceration through the stomach wall. Violent exacerbations of pain may be present during the active process of digestion.

The clinical picture presented by this group of tumors varies within the widest ranges from complete euphoria and utter unconsciousness of gastric changes to conditions fraught with grave consequences to health and life. This diversity of manifestations depends largely upon the size, location, and nature of the growth. Intramural growths, of small or even quite large size, located at a distance from the pylorus, may give rise to no appreciable symptoms. Large ones are prone to manifest themselves through their mechanical effect, producing a sense of epigastric weight and dragging. Frequently there are present dyspeptic manifestations of various degrees; loss of appetite, loss of body weight, anemia, and even pain. Such large growths are usually palpable through the abdominal parietes.

Where the growth is located in the pyloric region, and especially if it is pedunculated, it is more apt to be associated with active dyspeptic symptoms such as have just been mentioned. Epigastric or hypochondriac pains seem to be one of the most constant manifestations. A frequent tendency of pedunculated growths of all types near the pylorus is to prolapse into this opening and cause a partial or complete obstruction. This gives rise to very violent attacks of pain with nausea, and more or less protracted vomiting. Depending upon the degree of obstruction, there results more or less gastric distention, food retention, and peristaltic rigidity. Frequently there is blood in the vomited matter. The obstruction occurs through a ball-valve action and in most cases is only temporary, the tumor falling back into the stomach and a longer or shorter period of quiescence ensues, to be interrupted again by other attacks of acute obstruction, until finally death occurs or they are relieved by operation. The acute obstructive attack is often the first active manifestation of the disease. Cleghorn reports the occurrence of a pyloric perforation during an attack. This is very rare. More frequently there is intussusception. The stomach may be intussuscepted into the duodenum, as in a case recorded by Collier, or even the duodenum and part of the stomach into the jejunum, as in Wad's unique case. The tumor in Wad's case was a non-pedunculated fibromyoma near the pylorus.

When the tumor involves the mucous membrane, hemorrhage is a frequent symptom. It is due to excessive vascularity of the growth of the surrounding mucosa, or to the erosion of a vessel by ulceration or sloughing. The blood may be visible or

occult in the stomach contents or stools. As a rule even though profuse, the hemorrhage is without serious consequences. Launersh great multiplicity of goblet cells; such a finding, he believes, should always arouse suspicion of a polyposis. The diagnosis in



Fig. 8.

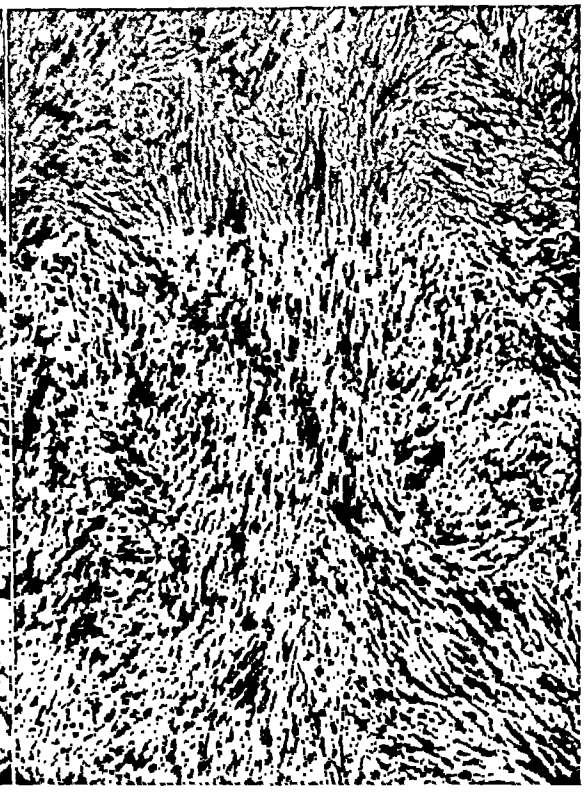


Fig. 9.

and Kemicke have reported cases in which it proved fatal. It is difficult to formulate rules of diagnosis regarding stomach content examinations aside from the period of acute obstruction. The majority of case reports include no data on this subject. From the few recorded cases and theoretical considerations, we may safely conclude that where the mucous membrane is not involved and there is no obstruction from the growth, normal conditions of motility and secretion should prevail. Where, however, the growth is one essentially of the mucous membrane, as in mucous polyps of the multiple as well as of the single types, achylia has been reported (Myer 1; Wegele 23; Chosrojeff, 23; Campbell, 23). Myer points out that the achylia is not an ordinary simple one, but is associated with excessive production of mucus, like egg-white, such as might be expected with several cases has been rendered possible by finding a tumor fragment in the vomitus, wash water, or feces.

ROENTGENOLOGY

The recent rapid development of radiological diagnosis, particularly the direct demonstration of abnormal conditions in the diagnosis of these tumors, is the best means short of the actual demonstration of the growth itself. I do not see how a diagnosis can be made of those tumors which are not palpable without the use of the *x*-ray and as all of us are thoroughly familiar with the importance of the *x*-ray it would be useless for me to elaborate on this common means of diagnosis.

CASE REPORT

Summary: Patient presenting no symptoms of stomach pathology excepting severe gastric

hemorrhage, one late in 1926, and one three weeks before operation; *x*-ray diagnosis of papilloma or cancer of the cardiac end and upper third of lesser curvature of stomach; at operation fibromyoma of stomach found and removed.

On June 17, 1929, Mrs. M. L., aged forty, No. 26948, entered the hospital because of hemorrhage from the stomach, weakness and for *x*-ray study.

Family History: Father died at the age of fifty-seven with apoplexy. Mother is living and well at the age of seventy-nine. The patient has two brothers and three sisters living and well.

Past History: The patient had scarlet fever and typhoid fever as a child; had an appendectomy and uterine suspension in 1922. She has three children living and well, aged twelve, eleven and nine, and no miscarriages. She has had frontal sinus trouble since 1928. Tonsillectomy was performed in May, 1929. In June, 1926, the patient contracted an acute cold; at this time she had considerable palpitation of the heart and three days after its onset, she had two severe hemorrhages from the stomach. The patient attributes this hemorrhage to some medicine she had taken for her pleurisy. Following this attack of hemorrhages, she was put on a Sippy diet for six weeks and did not have any pain, vomiting or discomfort and was able to take almost any sort of food. The patient was practically free from symptoms, except for a feeling of fullness and discomfort on pressure in the epigastrium. On June 2, 1929, she had an unusual desire to defecate and the stools were black. Later in the night of June 2, 1929, she vomited bright red blood with clots. This attack followed a considerable amount of physical exertion on the day and evening before. She was extremely exhausted and tired on the day of this hemorrhage. Her last menstruation was June 6, 1929, which was normal. There had been no recent loss of weight. Her habits were the very best; she does not drink, smoke or use tea or coffee.

Physical Examination: The patient was well nourished. Pulse 72; temperature 98; and skin very pale; blood pressure 108/70. The head had no abnormalities in shape or size; no growths or other irregularities. The teeth were in good condition. No glandular enlargements were noted in the neck; no abnormal pulsations; no enlargement of the thyroid. Chest: The heart was not enlarged. There was a slight mitral systolic murmur at the apex in the fifth interspace. Lungs: Careful examination revealed no abnormalities to inspection, auscultation, or percussion, there being no area of dullness; diaphragm excursions were normal on both sides; there was no retraction of any interspaces; no râles were heard over any portion. Genitourinary Tract: Negative. Extremities: These were normal in every particular, showing no atrophy, no loss of motion, no arthritis. Reflexes: Plantar, anterior tibial, knee jerk, abdominal and forearm reflexes were not exaggerated and were responding equally on both sides. Abdomen: There were no abnormalities in shape, size, or general contour of abdomen on inspection. No masses were palpable. There was an old scar in the midline below the umbilicus from an operation some years ago. Slight tenderness over the epigastrium was noticed on deep pressure.

Laboratory Report: Blood count June 17, 1929: Hemoglobin, 40; red blood cells 3,200,000; white, 6300, Polymorphonuclears 68 per cent, lymphocytes 32 per cent. After transfusion of 500 c.c. of whole blood, June 28, 1929: Hemoglobin, 60; R.B.C. 4,250,000; W.B.C. 8000, Polymorphonuclears 69 per cent, lymphocytes 31 per cent, Ind. 7.

Urinalysis was negative at all times.

X-ray pictures were taken of this patient during her first few days in the hospital and there was no evidence of pathology found by the fluoroscope in the erect position. However an examination a few days later in the reclining position showed a defect in the posterior wall of the stomach near the cardiac end which will be observed in the accompanying *x*-ray plates.

The site of these tumors is usually at the pyloric end of the stomach and the fact that this one was near the cardia makes the case which I am reporting interesting not only because of the location in the stomach, but because of the rarity of such tumors in this organ.

Operation was performed (procedure described in discussion).

Further History: The patient made an uneventful recovery. She did not vomit once after operation. The stitches were removed on the eighth day and in the early part of the ninth day, the patient had a spell of coughing and experienced a pain in the left lower quadrant, but the pain disappeared on the third day.

During these three days, her temperature was 102°F. She left the hospital on the sixteenth day after operation and now reports herself free from digestive disturbances.

Pathological Report: The specimen is a tumor mass, measuring 9.5 cm. in length by 5 by 5 cm. On one edge of it there is mucous membrane with some ulceration. The rest of the surface is rather firm and grayish with a yellowish tint indicating a fatty or degenerative process. Attached to this is a smaller tumor, measuring 6 by 3.5 by 2.25 cm. It is similar in form to the other and on section shows a cystic cavity, whose inner lining is rough with fibrous tissue strands running through it. Its contents were a clear mucinous fluid. This has the same general structure as the larger tumor. Microscopical findings: The section shows a neoplastic growth composed of fibrous tissue and smooth muscle cells. It is quite typical of bundles interlacing and some areas showing degeneration. There is no evidence of malignancy.

CONCLUSION

This case has been very instructive to me and has stimulated me to consider the possibility of the existence of benign tumors in obscure cases of gastro-intestinal disorders, associated with the presence of occult blood in the stools. It has also drawn personal attention to the fact that in operating upon tumors of the stomach, one should bear in mind that while benign tumors of this organ are rare, they are not so rare but that they should be considered in deciding upon a surgical procedure.

It is my hope that the readers of this article will note that the condition is not as frequent as it might appear at first thought and that cases that are unusual or are undiagnosed should be more thoroughly *x*-rayed and operated upon earlier.

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DISCUSSION

Dr. Harlan Shoemaker: The outstanding feature in this paper is the diagnosis by the roentgenologist, Dr. J. W. Warren. *X*-rays are taken, as a rule, in the prone position. Such an *x*-ray would have entirely missed this tumor of the stomach; an *x*-ray in the dorsal position very distinctly outlined the tumor.

Dr. A. A. Blatherwick: It was my privilege to assist Dr. Johnson in this operation. This diagnosis was made largely on the history of severe, recurrent hemorrhages, in the absence of the usual symptoms of carcinoma or ulcer of the stomach. The roentgenologic examination confirmed the diagnosis.

A short description of the technique of the operation may be of interest: After exposing the stomach in the usual manner, an incision about 3 inches long was made in an avascular area of the anterior wall of the stomach. The tumor, which was in the posterior wall, was everted through the incision in the anterior wall. An incision was then made through the mucosa, extending around the ulcerated surface over the tumor, an area about one inch in diameter. The tumor was then shelled out of its capsule in a manner very similar to that employed in removing a fibroid from a uterus. The serosa on the posterior wall of the stomach was not opened. Closure of the site from which the tumor was removed was accomplished by two rows of chromic sutures bringing the muscles and mucosa together. The incision in the anterior wall of the stomach was repaired and the abdomen closed without drainage.

Dr. Johnson (*closing*): I do not believe this tumor would have been discovered if the patient had not been put in a dorsal position, as the barium would not have collected around it because it was too high in the cardiac end.

PSEUDOMENSTRUATION IN THE HUMAN FEMALE*

CHARLES MAZER, M.D., F.A.C.S., AND A. J. ZISERMAN, M.D.

PHILADELPHIA

AS a preliminary to the discussion on the occurrence of pseudomenstruation in the human female, it is essential to review briefly the mechanism of the normal menstrual cycle.

As early as 1908, Hitschmann and Adler[1] demonstrated the cyclical changes in the endometrium of the normal human female; in 1913, Schröder[2] correlated these cyclical endometrial changes with the corresponding rhythmic phenomena in the ovaries.

According to our present conception of the probable mechanism of the menstrual cycle from recent experimental evidence, the rhythmic intermittency of female sex hormone production by the Graafian follicle and its successor, the corpus luteum, under stimulation from the anterior pituitary lobe (Smith[3] and Zondek[4]) is responsible for the cyclical endometrial growth and its subsequent dismantling in the form of menstruation. This assumption is supported by the experimental induction of menstruation in the castrated monkey by the administration of female sex hormone (Allen[5] and Morrell[6]). These experimental observations coupled with others soon to be mentioned seem to indicate that female sex hormone supplies the essential mechanism of menstruation. As will be seen later this form of menstruation lacks the progestational phase.

In addition to producing the female sex hormone, the mature corpus luteum elaborates a water soluble principle known as "progestin" or inhibitory hormone, the function of which is mainly to sensitize and prepare the endometrium for nidation and to inhibit further development of Graafian follicles. The secretory or premenstrual stage of the uterine mucosa, including the pseudodecidual reaction in the compact layer, is due to the latter hormone. This was first suggested by Fraenkel[7] and recently corroborated by Weichert[8] and Corner and Allen.[9] They proved the absolute dependence of the secretory phase of the menstrual cycle upon the hormone, "progestin," simultaneously produced by the corpus luteum.

The premenstrual endometrium differs from the interval endometrium in that it is thicker; the glands are cork-screw in appearance and distended with secretion; the nuclei of the columnar epithelial cells lining the lumen of the glands are eccentrically located; the blood supply is abundant; the stroma is edematous and contains numerous pseudodecidual cells.

Whether the rhythmic periodicity of anterior pituitary lobe function upon which ovarian function depends is inherent or is controlled by forces unknown to us is a matter of conjecture. Nor is it definitely known that the anterior pituitary gland produces two individual sex hormones, a follicle ripening (prolan A) and a luteinizing hormone (prolan B). In our experience, the luteinizing hormone (prolan B) now available is capable of ripening the primordial follicles of immature animals. This and other observations lead us to believe in the existence of only one pituitary sex hormone.

PSEUDOMENSTRUATION IN THE MONKEY

That cyclical uterine bleeding is always the culmination of maturation of the Graafian follicle and regression of its successor, the corpus luteum, following the failure of fertilization, was doubted by Heape[10] and later by Van Herwerden[11] as a result of their observations on preserved uteri of the Macacus rhesus. Corner[12] definitely established the occurrence of

anovular menstruation in this species as a non-seasonal event. He also showed that anovular cyclical bleeding is invariably associated with an interval endometrium, presumably because of the absence of the corpus luteum.

Further experimental proof that menstruation without ovulation and without the presence of a premenstrual endometrium is possible in the Macacus rhesus was furnished by Robertson[13] and others who observed uterine bleeding in the castrated Macacus rhesus after the withdrawal of injections of female sex hormone.

The observations by Heape, Van Herwerden, Corner and Robertson on the Macacus rhesus permit the following conclusions:

(a) Menstruation without ovulation is a regular occurrence in the Macacus rhesus during the non-breeding season; and that it is a non-seasonal event in a large number of the species.

(b) When an interval endometrium is found at the beginning of menstruation, there is a total absence of ovulation and corpora lutea.

PSEUDOMENSTRUATION IN THE HUMAN FEMALE

The sexual cycle of the Macacus rhesus duplicates that of the human female in time, periodicity and the occurrence of actual hemorrhage; therefore, comparison with that of our own species is scientifically sound.

The absence of ovulation and a corpus luteum in the last phase of the menstrual cycle was observed by some in the course of abdominal operations on regularly menstruating women, free from pelvic disease. Thus, a careful observer like Allen[14] says: "In one or two cases the evidence clearly points to absence of ovulation in the last several cycles. This condition, menstruation without ovulation, so common in the monkey, must be recognized as occurring in women."

An illustrative case came under our observation recently:

Mrs. R. R., aged twenty-three, and the mother of a two-year old child. Menstruation began at the age of eighteen, when her periods became delayed and somewhat irregular. She was admitted to the Mount Sinai Hospital on September 24, 1930, with a diagnosis of acute appendicitis and retroversion of the uterus. A lower median incision was made to permit also correction of the retroversion. She was menstruating at the time of operation. Both ovaries were somewhat enlarged and studded with numerous small cysts. The ovarian tunic of both ovaries seemed thick. Numerous punctures were made to evacuate the cysts. There was no evidence of either recent or old corpus luteum.

With these thoughts in mind, we investigated the microscopic appearance of the endometrium and, simultaneously, the blood-hormone content of a series of 41 regularly menstruating sterile women, whom we curetted a day or two before the expected flow and, simultaneously, abstracted 80 c.c. of venous blood to determine the level of female-sex-hormone by the Frank and Goldberger method.[15] For the sake of accuracy, we ran a duplicate test simultaneously in each case. Careful study of these women revealed no apparent cause for the existing sterility. The patency of the Fallopian tubes and proper insemination of the cervix had been previously established. Pelvic examination, basal metabolism tests and complete medical study had eliminated, as far as possible, local and constitutional causes for the existing sterility. They were therefore classed as cases of functional sterility. The results, as tabulated below, show that 24 of the 41 regularly menstruating functionally sterile women subjected to premenstrual curettage showed a non-nidatory endometrium, indicating defective luteinization or a total absence of the corpus luteum.

Women who suffer from menstrual derangements are either totally sterile or have a low fertility, but one hesitates to attribute an existing sterility to an endocrine cause in a woman whose menstrual cycle is normal, though there is no other ascertainable condition to account for the

sterility. This method of investigation is therefore helpful in the diagnosis of sterility in regularly menstruating women. A typical case history follows:

L. C., aged thirty-two, has been married twelve years and was never pregnant. She began to menstruate at the usual age but the intervals until marriage were somewhat prolonged. She is of average height and weight and presents no stigmas of an endocrine malfunction. Blood count, blood Wassermann reaction, urine and basal metabolism rate are normal. The uterine body is small and anteverted; the cervix long and narrow; the external os pin-point. There is no palpable pathology in the adnexa and no evidence of cervical infection. Well formed motile spermatozoa were found in the cervix four hours following intercourse. The Rubin test showed patency of the Fallopian tubes at the low pressure. Eighty cubic centimeters of venous blood, abstracted two days before her expected period and treated by the Frank and Goldberger method, produced no reaction in two castrated female white mice. The uterine scrapings, obtained simultaneously, showed an interval stage with areas of hyperplasia.

The failure to find a premenstrual endometrium and a demonstrable quantity of female sex hormone two days before the expected flow in a woman who has been menstruating regularly for twelve years is sufficient evidence of ovarian dysfunction —defective luteinization.

Table 1

Relationship Between the Condition of the Endometrium and the Level of Female Sex Hormone

Premenstrual endometrial findings	Female sex hormone level			
	Normal	Threshold	Neg.	Total
Interval endometrium	0	1	11	12
Hyperplasia	1	2	5	8
Premenstrual endometrium with local hyperplasia	0	0	4	4
Premenstrual	6	6	5	17
Total	7	9	25	41

Group I. In this series of 41 regularly menstruating apparently functionally sterile women, 12 yielded an interval endometrium a few days before the expected onset of the menstrual flow; in 8 we found a well-marked endometrial hyperplasia. Only 1 of the 20 in this group showed a normal level of female sex hormone in the blood; 3 showed only a threshold quantity; the remaining 16 showed no demonstrable quantity of the hormone.

Group II. In 4 of these women, we found a premenstrual endometrium with local hyperplasia. None showed a demonstrable quantity of female sex hormone.

Group III. In 17 women we found evidence of a premenstrual endometrium. Six of these showed a normal level of female sex hormone; 6 a threshold quantity; and the remaining 5 showed no demonstrable quantity of the hormone.

The significance of the absence of a demonstrable quantity of female sex hormone in the circulating blood a few days before the expected flow was stressed by Frank and Goldberger[15] and others. It assumes greater importance when a duplicate test is run simultaneously as in our cases, because in a large series of tests on normal fertile women, we obtained a positive reaction in no less than 94 per cent. It is therefore possible that even those women in Group III who showed a premenstrual endometrium but no demonstrable quantity of female sex hormone are also hypofunctioning.

This constitutes the first detailed report in the literature of the finding of a nonnidatory endometrium a day or two before the expected flow in regularly menstruating women. Passing mention of this observation has been made by one of us[16,17] in former publications. These findings suggest, by inference, the possible failure of ovulation and corpus luteum formation similar to the condition often found in the Macacus rhesus.

The findings in the first group substantiate the current belief that the balanced action of female sex hormone and the nidatory principle of the corpus luteum is the responsible factor in the preparation of the normal premenstrual endometrium.

They also explain the underlying cause for the existing sterility in these women and suggest rational therapy, namely, the administration of appropriate quantities of female sex hormone followed by the administration of lutein hormone during the latter half of the menstrual cycle. Unfortunately, the latter, so essential in the preparation of the endometrium for nidation, is not available for therapeutic use because of the meager source of supply. Female sex hormone, alone, is usually inadequate in the treatment of functional female sterility. Furthermore, the underlying cause of ovarian hypofunction is usually a lack of anterior pituitary sex hormone upon which the ovaries depend. Hence the treatment should, in most cases, be directed to the pituitary gland if clinical and laboratory findings point to an anterior pituitary deficiency.

Fortunately, we now have at our disposal potent products of anterior pituitary sex hormone, the dosage of which, however, has not yet been determined. The luteinization effect of this hormone has been established experimentally. The possibility of hyperluteinization with suppression of estrus through the use of excessive doses was also observed experimentally. It is therefore difficult to determine the exact dose necessary to increase luteinization without ill effects in the human female. In 3 of 15 women belonging to this class, we observed delay in the expected menstrual cycle from five to seven days after the administration of apparently excessive doses of the hormone. Clinically, the administration of 20 R. U. every other day during the latter half of the menstrual cycle has given us encouraging results.

We cannot explain the infertility of those women, in this series, who presented a normal endometrium and a demonstrable quantity of female sex hormone, on the basis of endocrine hypofunction. We feel that undetermined causes, such as sexual incompatibility, may be the causative factors.

SUMMARY

Amenorrheic and irregularly menstruating women are admittedly hypofunctioning; women who menstruate regularly and in whom there is no demonstrable cause for the existing sterility present a problem in diagnosis. In these women, a premenstrual curettage and the simultaneous determination of the hormone content of the blood by the Frank and Goldberger method are invaluable from the standpoint of diagnosis and treatment. The failure to obtain a premenstrual endometrium and a mouse unit of female sex hormone a day or two before the expected flow in regularly menstruating women is indicative of ovarian dysfunction, either primary or secondary to anterior pituitary deficiency.

For the following reasons we may infer that these women do not ovulate:

(a) In the human and the Macacus rhesus, the presence of a normal corpus luteum is invariably associated with a progestational endometrium.

(b) An interval endometrium in the Macacus rhesus obtained at the onset of the flow is invariably associated with an absence of ovulation and corpus luteum.

(c) In the course of abdominal operations on regularly menstruating women, who were at the end of the menstrual cycle, some surgeons occasionally found a total absence of a recent or old corpus luteum.

(d) The absence of a demonstrable quantity of female sex hormone in the circulating blood a few days before the ensuing menstruation also points to a possible failure of luteinization in view of the fact that 94 per cent of regularly menstruating fertile women show a positive reaction at this phase of the menstrual cycle.

The usefulness of organotherapy in these cases is limited because one of the two ovarian hormones, "progestin," so essential in the preparation of the premenstrual endometrium, is thus far not obtainable for therapeutic use. The luteinization hormone from the anterior pituitary lobe now avail-

able for therapeutic use gives promise of therapeutic results.

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SALT FREE DIET IN THE TREATMENT OF SURGICAL TUBERCULOSIS

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A SURVEY of the development of modern surgery shows clearly well-defined epochs. The aseptic era was followed by splendid technical developments. One anatomic complex of organs after another was conquered by surgical therapy.

The last field, thoracic surgery, could be perfected after Sauerbruch first applied his method of difference in atmospheric pressure. It is no coincidence that the same surgeon six years ago started with the research of a new problem in surgery: the diet.

The growing interest in physiological chemistry suggested a return from rather mechanical ideas to biology. Circumstances favored these events. Hermannsdorfer's experiments with acid and alkaline diet on wounds indicated the dietary influence on the healing process. Schade's findings on the chemical reaction of wounds seemed to confirm the clinical results. The acid reaction of wounds, the beneficial influence of ketonic diet on the healing process, the unhealthy aspect of wounds and slow formation of granulation during alkaline nutrition, all these facts outlined the experimental path to be followed. Gerson's dietetic therapy needed scientific investigation and received Sauerbruch's attention. Local conditions encouraged the work. The surgical clinic in Munich housed all kinds of tuberculosis patients. Lupus, surgical tuberculosis and phthisis were put under observation. Free from enthusiasm, even rather sceptically, did Sauerbruch and his staff start their work. Technical difficulties and numerous other obstacles had to be overcome. After a few months of pioneering they decided to investigate this problem from every possible angle.

Various theories existed about diet in tuberculosis. Nearly every-one of them was based upon the necessity of high caloric



FIGS. 1 and 2. (From Nissen-Myer_Ruegg.)

value, since Brehmer's experience had shown time and again how beneficial so-called "fattening diets" proved in cases of tuberculosis. At present Hermannsdorfer allows 3100 calories daily, whereas Gerson administers only 2560 calories. This difference is partly due to a variation in protein intake, which Gerson limits to 60–70 gm. per week, while Hermannsdorfer gives much more. Theoretically a small amount seems not to be adequate. MacCann and other authors showed that the minimum nitrogen content in tuberculosis patients is about normal or somewhat increased, namely 2.4 — 9.5 gm. per day. It is possible sometimes to activate the protein balance by giving only 1 gm. of protein per kg. body weight. Gerson's small amount of protein is therefore even somewhat dangerous over long periods.

Hermannsdorfer and Gerson both allow carbohydrates in restricted amounts. Looking at this fact from a general viewpoint it seems rather difficult to under-

stand. We know that the French theory of usually severe dysfunction of the liver in tuberculosis is not justified. The levulosis test proves normal or almost normal liver function in tuberculosis patients. Carbohydrates are easily absorbed, have little or no dynamic effect, improve assimilation of fat and have an economical value for proteins. For these reasons Monceaux favors a large intake of carbohydrates and claims furthermore that they improve the detoxicating functions of the liver. The difference in carbohydrate estimation is evident if we compare the old opinion to the Gerson-Hermannsdorfer diet. An explanation will be given later.

Fats are allowed in abundance, a rather natural procedure. In addition cod liver oil is given combined with phosphorus. The value of phosphorus seems somewhat problematic. Straub's investigations showed that the phosphorus content in Hermannsdorfer's prescription varies very much for technical reasons so that the importance of phosphorus seems doubtful.

Much more interesting in fact is the main part of the diet: strict lack of salt. This rule was sternly followed by both Hermannsdorfer and Gerson, and sustained in spite of later changes and disagreements. There have always been different opinions about the role of salt in medicine. Our body really does not require much salt. There is no protein known which contains chlorine ions. Hydrochloric acid of the stomach is reabsorbed by the intestines and returns to the stomach. The regulation of osmotic conditions, the main work of NaCl in the body, is guaranteed by a very small amount of salt; 2 gm. daily are sufficient for our organism, a dosage which ordinarily is contained in food. Even this amount is already high compared to other mineral salts. Artificial increase of salt must cause an overbalance in the system and unnecessary work in elimination and storage.

Our way of cooking spoils the natural taste of food and makes additional spicing necessary. There is no substantiated reason for the opinion that a higher amount of salt is necessary than naturally provided in foods. We know people who practically do not use salt at all; and Hermannsdorfer and Gerson could demonstrate on many patients that lack of salt is not harmful.

Tuberculosis patients are comparatively poor on NaCl. Hermannsdorfer considers this as a self-defense of the organism and wants to help the body by future restrictions. He also claims that salt retains fluid. Deficiency of salt consequently diminishes the fluid and at the same time discharges from fistulas, granulations and so on. Anyone acquainted with this diet knows this change very well. Doubtless the lack of salt is responsible for this striking effect. Meyer-Bisch claims that a salt-free diet has not always a dehydrating effect and might produce NaCl retention; this observation certainly cannot be generalized.

But which one of the two ions in NaCl is the undesirable one? Recently the effect of Cl ions was found to be harmful. Keining and Hopf made interesting studies and now hold the Na ion responsible for disturbances in the chemistry of mineral salts.

Studying the role of NaCl in connection with the metabolism of all the mineral salts in the body, we have to deal with many difficulties. Demands, distributions and relations of the various inorganic salts depend on a multitude of organs in our body. Endocrine glands and the reticulo-endothelial apparatus play a role besides the nervous system.

Our skin is rich in mineral salts. Luithlen was the first to consider salt harmful in skin diseases. The Hermannsdorfer-Gerson diet gives excellent if not its best results in the treatment of lupus. This fact seems to prove the importance of NaCl and mineral metabolism in this diet. Robin's theory of demineralization was later corrected by Monceaux. He observed differences according to pathological-anatomical varieties and stages. There is a marked deficiency during the acute stage which becomes more balanced in the chronic course. The output of mineral-salts decreases as the organism tries to hold back these valuable

substances. An abundant intake of mineral salts is important because they are stored again in a slightly active balance already. In the final stage of the disease the body apparently loses its economical ability and the output increases. Gerson's idea to sustain the natural demand by administering mineralogen seems quite appropriate. Mineralogen contains 10 different mineral salts. Of course, it is true that large amounts of vegetable and fruit provide enough of these substances, and it is also true that some authors obtained the same results by Gerson's diet without mineralogen. But certainly no harm is done by this mixture, and if the body has no use for these substances it eliminates them.

Another reason for restriction of salt and changing to mineral salts is the so-called transmineralization which stimulates the system (Luithlen, Wiechowski). The sodium in the diet is extensively replaced by calcium, magnesium and strontium which confirms Keining and Hopf's aforementioned theory to hold the sodium responsible for ill-effects of salt. But sodium can be neutralized by administration of a salt which contains the minerals exactly in the same percentage as found in human serum. Titro salt is said to be effective in all diseases connected with the sympathetic nervous system. The use of titro salt in the Hermannsdorfer-Gerson diet could eliminate many technical difficulties. Evidently Hermannsdorfer does not consider this possibility. He finds it some what illogical, according to a private remark, to give a poison and its antidote at the same time. He prefers to omit it.

The diet is very rich in vitamins. To substitute phosphorus cod liver oil by vigantol is possible, but very exact dosing is necessary because an excess may produce bad effects.

Almost every article concerning this diet, and there are many, expresses a separate opinion on the chemical reaction of this regime. It may be mentioned again that the favorable effect of acid diet on wounds has been the fundamental idea for the combination of this diet. Hermannsdorfer's diet in its last form undoubtedly has a lower pH value than Gerson's who restricts meat more and more. He is nearing a complete vegetable fruit regime. R. Berg and others also claim an alkaline reaction of Hermannsdorfer's diet. The same discrepancy exists on the chemical action in the body. It must be said again that there is a great difference between a chemical reaction and the physicochemical reaction in the body. What tastes sour to us may be alkaline for the body and vice versa. Fruit contains fruit acids, but they are precipitated immediately and the alkaline components dominate.

Hermannsdorfer bases his opinion of the chemical reaction of his diet on urine examinations, but it remains questionable if this method furnishes useful results. The importance Sauerbruch gives this question may be best expressed in his own remark at the 1930 surgical congress: "I do not even know what pH really means."

This attitude sometimes can be applied in medicine. There are empirical items lacking theoretical base which give striking results, so from this point of view the diet certainly proved its therapeutic value.

The best results are obtained in lupus. Hermannsdorfer goes so far as to suggest treatment of one case of lupus wherever his diet is given in other kinds of tuberculosis. If the lupus case is cured, the preparation of the diet is done correctly.

The value of the diet in cases of tuberculosis of the lungs is still much discussed, but the surgical form shows very good results. Fistulas of soft parts discharge less after a short time, the discharge becomes more serous and finally ceases while the fistulas close. Figures 1 and 2 show tuberculosis of the fingers before and after dietary treatment which the patient received at Sauerbruch's clinic. Tuberculosis of bones reacts likewise. Swelling of joints is reduced and even severe joint cases with old fistulas were cured. It is important that patients over forty-five years of age who usually give an unfavorable prognosis are very much benefited by the diet. I remember a man

forty-eight years old with tuberculosis of the hip joint and mixed infected fistulas who was treated in Munich during the early stage of the dietetic studies. The man was completely cured after all other therapies had been tried in vain. In 1926 Krecke at a surgical meeting reported the case of a ten-year-old boy with tuberculosis of the small foot joints. He was cured after eight weeks. Of course, these cases are not incurable and usually get well after eight to ten months, but the diet shortens the illness considerably. Since then a great number of authors have reported favorable results in surgical tuberculosis. Today we are entitled to include certain kinds of tuberculosis of the lungs into the surgical field. Phthisis, the emaciating disease, certainly represents a special type of tuberculosis and we cannot expect the same results as in cases of tuberculosis of the hip joint. Especially lung specialists in sanatoriums do not share Hermannsdorfer's optimistic opinion and remain rather skeptical. But it can be safely stated that the diet brings a considerable number of far-advanced and so long inoperable cases into the stage where surgical treatment can be applied. When we consider that according to Sauerbruch's statistic 67 per cent of operated cases resume their work, the value and results of the diet should not be overlooked or underestimated.

It happens very often in experimental medicine that two authors disagree on the same subject, mostly because conditions for their experiments were different. Many things have to be considered when it comes to a diet. Sometimes the original rules are not followed. If the mineralogen is omitted or its substitutes changed, no great harm is done. But if three meals are given instead of seven I think one of the main points is neglected. It is evident that digestion and food value are largely increased by frequent meals consisting of smaller portions, much more so than by three ample feedings. Sometimes reports are given covering only a period of a few weeks. Needless to say no real opinion can be formed in such a short time. It takes about two weeks to get the patient used to the diet. There are only a few who cannot stand the lack of salt, but this part of the diet represents most of the technical difficulties and requires a well and specially trained dietitian. This diet cannot be prepared in the usual way, therefore a special nurse or cook will be necessary. The surgical clinic in Munich started to get good results only after a special dietitian from Bielefeld (Gerson's place) was obtained and put in charge of preparing the food.

To cover up the lack of salt is less difficult in America than in Europe, because here more substances are available to give the food good taste. The all-year-round selection of fresh vegetables and fruits present another great advantage. But still the rule stands that a well-trained person has to be responsible for the food preparation. In New York about eight nurses are specially trained for this diet and they should represent a nucleus for future development. This complicated diet easily introduces misunderstandings and consequently bad results. Here another point must be mentioned. It is wise and very helpful to isolate patients on this diet and have them well watched. I remember in Munich, that during the early stages of this diet patients were kept in the same building with others. Nobody suspected or watched their fellow patients or visitors. The results obtained then were far less favorable than later on with isolation and strict control of visitors. We must not forget the psychology of the patients and the good-hearted visitors who will supply additional food, misled by their feelings.

In the course of the experiment Hermannsdorfer and Gerson advised different types of food. To avoid complications in selecting the special diet I mention for various reasons Hermannsdorfer's last publication. The cook book he has published in the third edition seems to give the diet which best represents the results of Sauerbruch's and Hermannsdorfer's experiences.

OS INTERMETATARSEUM AND HALLUX VALGUS*

PHILIP H. WHEELER, M.D.

CLEVELAND, OHIO

THE interest which prompted this review of *x*-rays of feet in approximately 500 persons was initiated by the discovery of an os intermetatarseum in a foot with hallux valgus which was *x*-rayed preoperatively. In this case it seemed that such a bone might mechanically aid in the production of such a deformity. This has been suggested by Köhler as a cause contributory to hallux valgus. With this in mind all foot plates in the files of the Lakeside Hospital for the past five years were examined to learn if this bone, easily overlooked, were present in other cases of hallux valgus. Five hundred and twenty-one plates permitted the ascertainment of the degree of valgus and the absence or coincident presence of the os intermetatarseum demonstrable by *x*-ray. Forty-two other plates were of feet with ununited epiphyses and are not included in this series.

Those feet showing the presence of hallux valgus, as determined by the degree of angulation of the first metatarsal and the proximal first phalanx and by the displacement of the sesamoid bones, were roughly graded as moderate, severe and very severe valgus, it being taken into consideration that few so-called normal feet of those examined showed a perfect alignment of the metatarsal and phalangeal bones. With such a classification 26 per cent (138) of the feet, including 23 pairs with double valgus and one pair with unilateral valgus, were classified into 60 per cent (84) with moderate valgus, 30 per cent (42) with severe valgus, and 10 per cent (12) with a very severe valgus. In the total series, however, the os intermetatarseum was found but 24 times. In 63 per cent (15) valgus was associated with the os intermetatarseum. Thirty-seven per cent (9) of the cases with this bone present showed no evidence of valgus.



FIG. 1. Bipartite os intermetatarseum.

This leads one to believe that while the os intermetatarseum may produce some tendency to valgus, the latter is by no means always associated with the presence of this bone.

Conclusions may need to be somewhat tempered in the light of the findings of the anatomists who report an incidence for this bone of about 8 per cent, whereas we have been able by *x*-ray to demonstrate but 4.6 per cent. It may be that our failure to visualize it more frequently is due to its cartilaginous or poorly calcified nature. Such a presumption is in accordance with the findings of those who have made dissections. Many plates were classified as not showing this intermetatarsal bone when a poorly defined area of increased density was evident between the bases of the first and second metatarsals, because this is the region of the dorsalis pedis artery which might have cast shadows

* From the Department of Roentgenology, Lakeside Hospital. Submitted for publication November 2, 1931

such as those seen and which in some plates could be identified as a sclerosed vessel. Only those plates which were examined several times at distinct intervals and which gave strong evidence for the presence of this bone were recorded as showing the os intermetatarseum. Other larger and better calcified bones of the foot were noticed in an incidence very close in percentage to that reported by anatomists from their dissections.



FIG. 2. Bilateral os intermetatarseum.

In this series with 26 per cent (138) of the plates demonstrating valgus, but 2.8 per cent (15) show in association with the valgus an os intermetatarseum, which has been suggested as a contributory cause of valgus. The additional fact that 37 per cent (9) of these feet with os intermetatarseum do not show valgus makes one feel that no great stress should be laid on this accessory bone as a contributory cause of foot deformity. Of those feet showing coincidental appearance of os intermetatarseum and hallux, 53 per cent (8) were grouped as moderate, 25 per cent (6) as severe, and 7 per cent (1) as very severe. Therefore, in addition to a frequent lack of association of valgus and os intermetatarseum, we may further say that the presence of an os intermetatarseum in a foot with valgus does not seem to make the degree of deformity unusually severe.

Two of the variations of os intermetatarseum reported by anatomists, but very uncommon in *x*-ray literature, were noted. They are fusion of the os intermetatarseum with the first cuneiform and fusion with the first metatarsal. A bipartite os intermetatarseum was discovered (Fig. 1). No previous report of a bipartite os intermetatarseum has been found in the literature. In 2 cases (4 plates) of 24 plates the os intermetatarseum was found to be present bilaterally. Köhler reports having seen one such case. In one pair there was associated a severe and a very severe hallux valgus. (Fig. 2.)

CONCLUSIONS

1. There is little evidence for the so intermetatarseum's contributing to either the presence or the degree of severity of hallux valgus.

2. A case of bipartite os intermetatarseum, hitherto unreported, is demonstrated.

3. Two cases of bilateral os intermetatarseum are reported.

[For References see p. 344.]

NEW INSTRUMENTS

THE BRUNET-FLAGGE NEEDLE DRIER

P. W. FLAGGE, M.D.

HIGH POINT, N. C.

TO date all attempts to dry hypodermic needles and like instruments satisfactorily have failed or have been too time-consuming to be practical. A persistent effort in this direction is conclusive evidence of the desirability of an instrument designed to accomplish this end.

When the needle is attached to the Luer syringe, a dead space is left, for mechanical reasons, between the extreme end of the Luer syringe slip and the inner end of the needle conus. This space always retains a portion of either the medication, cleansing or sterilizing fluid, and is not dried with any of the needle driers. Regardless of the kind of steel used in making the needle, or the character of the sterilizing fluid, ultimate destruction of the metal or occlusion of the lumen is the final result.

The importance of a clean needle free of erosion or a nidus of foreign material in any part of the shoulder or needle is appreciated by every user of hypodermic medications. Especially is this true in laboratory or intravenous work. Nothing is more exasperating or embarrassing than to have a needle in a vein blocked by a rapidly formed blood clot or an eroded surface either in the shoulder or the inside of the needle.

To overcome this defect in the construction of all driers on the market the form here described was designed. The improvement over all other driers consists in a provision whereby a backward flow of air effectually dries the shoulder and the inside at the same time. This backward flow of air is induced by grooves cut in the Luer tip of the drier which, together with the inner surface of the hub of the needle,

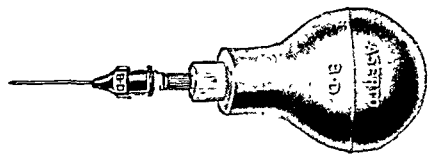


FIG. 1.

form channels which permit both a backward and a forward flow of air, thus drying the dead space at the end of the Luer tip and the inside of the needle at the same time.

The illustration shows how the air may be provided by a rubber bulb fitted on the stepped shoulders of the drier. A dry piston syringe may be substituted for the rubber bulb by changing the construction of the drier to fit the nipple of the syringe.

In practice the drying is effected by throwing over the end of the needle and hub a piece of gauze which catches the droplets and prevents them from being sucked back into the needle as the bulb expands. A few quick thrusts of air are sufficient to dry the needle and leave it with a clean surface.

This drier is small enough to be enclosed in the usual hypodermic unit.

Until such time as needles become so cheap that they may be discarded after a single service, this simple device will pay for itself many times in the course of a year.

A NEW TOURNIQUET*

H. DAWSON FURNISS, M.D., F.A.C.S.

NEW YORK

THE tourniquet here illustrated has been used by me, and friends to whom I have given it, for several months with the greatest satisfaction. It consists of two metal parts and a strip of cotton elastic, ¾ inch wide and 18 inches long. The friction portion, that on the right, is hooked over the tongue-like part of the other metal piece. By traction on the free end of the elastic any desired degree of tension may be obtained. To relieve tension the round metal tab is lifted lightly toward the left, which allows the elastic to run through the friction portion. It has the advantage of allowing tension to be increased evenly, without jar, and either slowly or rapidly. It is much more comfortable than rubber tubing, and there is less chance of pinching the skin.

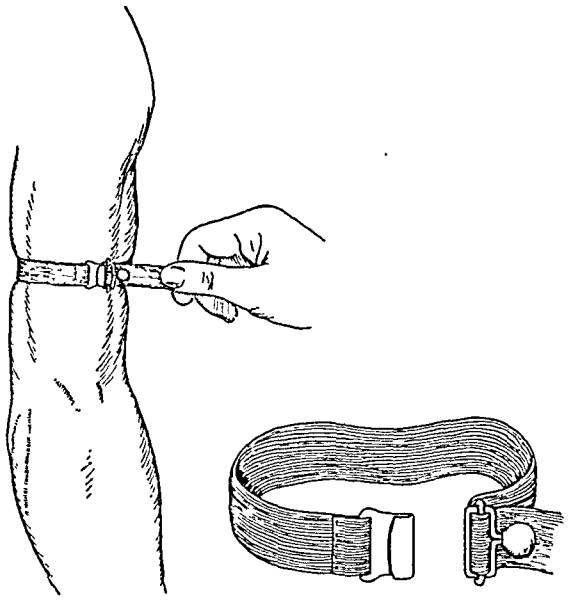


FIG. 1.

The device, originally intended for holding papers, attracted my attention at the Business Show. To make the tourniquet, the only change necessary was to substitute elastic for the webbing.

* Submitted for publication January 14, 1932.

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EDITORIALS

MEDICAL ECONOMICS

THESE are days when a majority of physicians have an interest in the economics of medicine. Ways and means of balancing the budget are favorite subjects in hospital board discussions. At a recent conference of hospital superintendents the presiding officer asked those present to raise their hands if the hospital they conducted had met expenses during their last fiscal year. The response was less than 5 per cent. We do know the physician has been no exception in that his income has suffered a marked reduction. Following the financial trend his real estate holdings have decreased in value and his savings in the form of stocks and bonds have almost reached the melting point. Yet there has been no complaint. The man engaged in business or on a salary wails about his cut in pay but, for the most part, the physician has remained silent. That he has been and is financially affected is no secret.

At a medical gathering this topic casually crept into the discussion. Part of the talks were devoted to certain abuses. One was that the law should be changed to read that only American citizens or those who have legally signified their intention of becoming citizens be granted a license to practice medicine. It was pointed out that we permitted anyone who could pass a State Board examination to practice. Inasmuch as economic conditions in foreign

countries are very bad and as this country is looked upon as a land of gold and milk and honey, in spite of our economic upheaval, all who can reach our shores do so, and shortly are in competition with our medical citizens. Needless to say all did not subscribe to this reasoning. Another of the group suggested that only graduates of medical schools in this country be permitted to practice. Stories were told of American physicians who went to France during the war, married French girls, decided to remain and practice and did so, after they had spent another four years studying medicine in France. To this view there were dissenting opinions. Another gentleman attacked what he called "the visiting teacher racket." A physician from a foreign clime, usually of more than the usual attainments, finding it hard to make ends meet in his mother country, by various means gets invited to this country to give a course of lectures in one of our medical schools. He arrives with great fanfare and flourish of trumpets. Usually he is well press-agented. He gives a few lectures and talks before medical groups and lay organizations. Meanwhile, he opens an office and for eight or nine months of the year engages in active practice, assiduously devoting himself to making money. Being connected with a teaching institution he is not required to pass a State Board examination (at least, this is true in New York State). It was conceded that it was fair and proper for any foreign physician to come here and lecture, but that he should give his lectures, be paid for giving them, and then call it a day. However, as one of the group said, "Only a few are invited so why even consider the subject?" The matter of free and part pay clinics came up in the discussion, but this is an ancient topic and needs no further mention.

Naturally, nothing was settled. Out of it one would conclude that many physicians know or feel something is wrong with our economic structure but look to the other fellow to correct the faults that affect their means of making a livelihood.

We are interested in learning your opinions concerning these problems. It may be you harbor resentment about many things we have not mentioned. You may think all is well with us and that the future is secure. Perhaps you agree with the advice, once given, "Stick to your task, do good work, and the income will take care of itself."

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CORRESPONDENCE

To the Editor:

Your article in the August number of THE AMERICAN JOURNAL OF SURGERY concerning Dr. John Fleet was of particular interest to me. I happen to be descended from John Fleet's sister. I know of no portrait in our branch of the family of John Fleet. He had a nephew, John Fleet Eliot, who died in Boston about 1885 and left his manuscripts and papers to the Massachusetts Historical Society. If he had a portrait of him it would be found in the archives of that Society.

There are in the possession of my Mother two leather fire buckets that belonged to Dr. John Fleet. At that time every citizen in the city of Boston was required to have in his front hall two fire buckets and was required to answer all fire alarms. I am sending you a photograph of these buckets, which I thought might be of interest to you.



FIG. 1. Fire buckets which belonged to Dr. John Fleet of Boston (1766–1813), now in possession of his great-niece, Mrs. Katherine Alden, Los Angeles.

ELIOT ALDEN, M.D.

Hollywood, Calif.
August 22, 1932.



AMERICAN PHYSICIANS

JOHN REDMAN COXE

JOHN REDMAN COXE, born in New Jersey in 1773, received his education in Philadelphia under his grandfather's care, until, at the age of ten years, he was sent to England, where he resided for seven years. He completed his classical education in Edinburgh, where he also attended a course of medical lectures at the university.

Returning to America in 1790, he studied medicine under Dr. Rush. He graduated from the University of Pennsylvania, after which he again went to Europe and visited the hospitals of London. Later he studied in Edinburgh and Paris. In 1797 he again returned to Philadelphia, where he began to practice. He became one of the resident physicians of Bush Hill Hospital, under the charge of Drs. Physick and Cathrall. He was appointed physician to the Port of Philadelphia in 1798, and to the chair of Chemistry in the Medical Department of the University of Pennsylvania. In 1819 he was transferred to the chair of Materia Medica and Pharmacy. He was elected to the medical staff of the Pennsylvania Hospital in 1802.

Dr. Coxe was at one time the editor of the *Medical Museum*. He published as editor "The American Dispensary," in 1808, a medical dictionary, "Exposition of the Works of Hippocrates," and an "Essay on the Origin of the Discovery of the Circulation of the Blood."

Dr. Coxe was an advocate of vaccination. This was illustrated by the fact that he vaccinated his child, then an infant, "at a time when the full efficacy of the practice was still in suspense in the public mind. He fully tested it, by exposing him [the child] to the influence of the smallpox after vaccination; the result of this bold experiment contributed to establish reliance in the protective power of vaccination."

He died March 22, 1864, at the age of ninety-one years.

T. S. W.



JOHN REDMAN COXE

[1773-1864]

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BOOKSHELF BROWSING

THE ROLE OF EXPERIMENTS ON ANIMALS IN THE TREATMENT OF DISEASE*

CONTROL OF ANIMAL DISEASES

VERANUS A. MOORE

ITHACA, N. Y.

A GENERATION has passed since the experimental method was introduced into the study of microorganisms in their relation to disease by Pasteur and his compeers. It has been referred to as the "new pathology" because it associated intimately the causative factor and the tissue changes. It is not too much to claim that since the application of this method the results obtained have done more than all the observations of preceding centuries to raise human knowledge of animal diseases from "conditions of vagueness to conditions of exactness." The benefits derived from researches, since the adoption of the experimental method, can be understood only by comparing them with the results obtained by other methods in earlier times.

It is difficult to visualize the situation that existed seventy and more years ago when communicable diseases often decimated flocks and herds; when their cause and means of dissemination were shrouded in mystery; when the frantic efforts of owners to save their animals from merciless plagues were useless; and when medical sciences were as ineffective in explaining the cause of the diseases as the owners were in controlling them. In addition to the physical suffering of animals, there is the financial loss which not infrequently brings privation and want to many people. When animals that constitute the chief source of income in a community are destroyed by disease, the consequences become a matter of much public concern.

Data are incomplete on the extent of losses caused by infectious and epizootic diseases prior to the introduction of the theory of a specific etiology for the plagues of man and beast. Up to that time the cause of communicable diseases was believed to be outside the power of man to control. As a result, these maladies were allowed to spread according to the laws that govern their dissemination. A distinguished English veterinarian placed the losses of his country from 1839 to 1870, inclusive, due to rinderpest and contagious pleuropneumonia of cattle alone, at $400,000,000. It was the terrible havoc wrought by these diseases in Germany that prompted the establishment of the *Tierärztlichen Hochschule* in Berlin in 1790, with the objective of finding some way of stopping their

* This is the seventh of a series of articles on the value of animal experimentation in medical progress. The next article will appear in an early issue.

ravages. In America, communicable diseases of live stock were beginning to appear in serious proportions at the time the Land Grant colleges were founded in 1862. It was one of their functions to investigate animal diseases. Today all but one of the eleven veterinary colleges in the United States are integral parts of state or Land Grant colleges, and the one exception is a school in an endowed university. The Federal Bureau of Animal Industry was established in 1884, for the specific purpose of officially combatting epizootics, and later state live stock sanitary boards were created. The function of these organizations was to study and control the communicable diseases of animals which threatened the animal husbandry of the country.

There are several diseases of live stock in the United States that formerly exacted annually a toll of many millions of dollars worth of animals which in recent years have been brought under almost complete subjugation. When owners become more familiar with the nature of these diseases and with what modern research has taught can be done to prevent them, the damage which they occasion will be reduced to a minimum. There are two important factors involved in the eradication of these maladies; namely, more definite knowledge of their nature, that can be attained only by continuous researches and experimentation, and the application of the knowledge acquired which requires further education of the agricultural population along these lines.

An understanding of the functions of the body and its nutrition has been the basis of successful methods for improving the care and handling of animals. This is not restricted to their physical comfort, in the surroundings in which they must exist, but it applies equally to their productiveness. It is natural that those interested in the improvement and higher development of animal husbandry and the industries depending on it should desire, not only to retain the advantage they have acquired already, but to increase the benefits through further researches and study. As the conditions under which animals are kept vary, so the diseases from which they suffer undergo appreciable changes. This means that to safeguard animal husbandry, which is the greatest industry in agriculture, the new and constantly changing problems associated with it require renewed and continuous study.

Further objectives in animal experimentation are to ascertain additional facts concerning their physical requirements whereby the methods of handling them may be improved in such a way that better health may be insured and untimely death from disease prevented. One need but glance at the methods of handling farm animals a century ago and then witness the conditions under which they are kept today to appreciate the improvements that have accrued from the experiments that have been made on subjects pertaining to sanitary and otherwise healthy abiding places for farm animals. Further, their productiveness has been increased tremendously. The average quantity of milk per cow in dairy herds has been increased nearly, if not quite, 100 per cent. Again, better knowledge of the conditions responsible for the general diseases among animals points to more efficient methods of preventing them. Methods that are successful in controlling epizootic and other infectious diseases are, without exception, based on facts obtained from experimental work, and by no other means.

At times the diagnosis of an infectious disease requires the use of animals. Some years ago a disease appeared among the cattle in a number of herds in northern New York that resembled foot and mouth disease, and which was declared by some people to be such. The accuracy of the diagnosis was in doubt. The live stock sanitary officials were forced either to accept the diagnosis and destroy all of the animals on the farms where the trouble existed and indemnify the owners; or prove that it was not foot and mouth

disease. The experimental method was applied. Several healthy calves that had not been exposed were inoculated from the infected animals and the trouble was found to be a form of inflammation of the mouth and not foot and mouth disease. This simple experiment settled the points at issue in the diagnosis, saved the State from the stigma of having to record again the appearance of that disease, and saved the animals for their owners.

The extent of the losses formerly sustained from communicable diseases among animals is not recorded. It is known, however, that soon after the introduction of the experimental method the federal government estimated the loss in the United States from these causes to exceed $200,000,000 annually. The history of several of the more destructive diseases shows that they baffled all attempts at prevention until their specific nature was determined. Following the discovery of the cause of anthrax by Pollander, of tuberculosis by Koch, of glanders by Loeffler and Schutz, of septicemia hemorrhagica by Kitt, of Texas fever (southern cattle fever) by Smith, of infectious abortion in cattle by Bang and of hog cholera by de Schweinitz, a large amount of work was initiated to ascertain how each of these and other diseases were disseminated and by what methods they could be prevented. Where there are competent veterinarians, and live stock sanitary officers animal husbandry is protected against serious losses from these maladies. The great live stock industries of this country exist because of the application of knowledge obtained from carefully conducted experiments on the nature of the particular diseases that threatened their existence. Without this knowledge, the control of the great scourges of animals, like anthrax, contagious pleuropneumonia, foot and mouth disease, hog cholera, rabies, Texas fever, and many others, would be impossible.

The stabilization of animal husbandry, and the industries dependent on it, is due very largely to the control of infectious and epizootic diseases that for centuries barred its progress. The success attained is due to results of experimental work. To understand fully what this method means by way of elucidating the nature of disease, as well as exemplifying the economic significance involved, one must have a clear perspective of the individual results of research that have been obtained. A brief summary of the history of the more important infectious diseases of live stock will portray more clearly, perhaps, than any other statement our indebtedness to the experimental method.

ANTHRAX

Anthrax has been known since very early times. It figures largely in the history of the early and middle ages as "a devastating pestilence attacking animals and through them mankind." Primarily it is a scourge of cattle and sheep, but often of other animals and man. Cruickshank states that it caused annually the death of fully 500 people among the wool sorters in London. Prior to the discovery of its cause thousands of sheep and cattle died of it each year. When its nature was revealed, the means by which it spread determined, and a vaccine produced, its ravages were checked. The method of vaccination, introduced by Pasteur, removed largely the dread of it from the mind of cattle and sheep owners of France. There is a no more spectacular event in medical history than the experiments made by Pasteur at Pouilly-le-Fort in demonstrating the efficiency of his vaccine to immunize cattle and sheep against anthrax. Not one of the vaccinated animals developed the disease after exposure, while all of the controls died. The vaccine enabled farmers to keep live stock in many places where otherwise it would not have been possible because of the prevalence of anthrax spores in the soil. This was especially true of large sections of France, Germany and Spain. Chester, at the Delaware Experiment Station, prepared a vaccine against anthrax that made it

possible to utilize for grazing lowlands along the Delaware River that previously could not be used because of heavy anthrax infection.

In 1899, Sobernheim discovered a method of vaccination consisting of an injection of anthrax bacilli with a quantity of immune serum. This is known as the simultaneous method, and it was, and still is, employed extensively by sheep raisers in South America. It is reported to be giving excellent results. Eichhorn states that immune serum is not only a valuable prophylactic against anthrax in animals, but an efficient therapeutic agent for the disease in humans. This is confirmed by a number of physicians who emphasize its value as a remedial agent. Pellon reported 205 cases among the industrial workers and their families in Marseilles, with but 25 fatal cases. The pronounced low mortality is attributed by him to the use of anti-anthrax serum. Jacobson has published on account of 61 cases in New York City that occurred from 1914 to 1923 of which 23 were caused by infection from shaving brushes.

Anthrax bacteria have been brought to many places in the United States with infected hides, wool and hair. Were it not for the preventive measures that are being applied wherever and whenever it appears, anthrax would soon become a serious menace to the sheep and dairy industries of the country. This is illustrated by outbreaks that have occurred at different times during the last fifty years. In 1906, it appeared in northern New York and spread to the animals on eighty-four farms. There were 170 fatal cases before the 3000, or more, animals in the locality could be immunized by vaccination. When that was accomplished, the disease disappeared. Since that time, it has been kept under perfect control by means of vaccination.

Further, anthrax is a menace to human life. The records show that there are approximately 130 cases in the United States annually. It is an achievement of much significance that a serum has been made with high therapeutic value that is reducing the mortality from a very high to a very low percentage. The proper use of vaccine and serum as prophylactics is decreasing steadily the number of outbreaks among animals which means a smaller number of human cases. The chief sources of human infection are diseased animals and infected wool and hair that are handled, and shaving and other brushes manufactured from bristles or hair that came from anthrax diseased animals.

The preparation of anthrax serum, testing the efficiency of the vaccines and making diagnoses, require the use of a few horses and experimental animals, but anthrax is no longer a serious menace. Many American farmers who live in infected areas have become so thoroughly accustomed to the prophylactic use of vaccines that they apply them as a recognized part of their routine operations. In this way thousands of acres of infected land are utilized for grazing purposes that otherwise would be idle. The only disease of animals, worthy of mention by the classical writers and the supposed murrain of the Book of Exodus has been brought under control by patient, painstaking experimentation.

CONTAGIOUS PLEUROPNEUMONIA OF CATTLE

Contagious pleuropneumonia originated in the highlands of Central Europe. It spread to every cattle-raising country in the world. It was estimated that during the first quarter of the nineteenth century it cost England and her colonies $450,000,000 in deaths alone. It was brought to the United States in 1843 in a cow imported directly from Europe and taken from shipboard to a Brooklyn cattle shed. Because there was little traffic in cattle, it spread slowly in the beginning. In a few years, however, it became widely disseminated in the Atlantic states. By 1880, it was evident to those most familiar with it that if it were not eradicated it

might, and very likely would, through some one of the increasing avenues of transportation, reach the cattle on the western ranges, where its control would be especially difficult, if not impossible. An important industry was threatened with impairment, if not extinction. Beef and dairy products, valuable and highly prized foods, seemed on the verge of soaring in price to heights unattainable by common people.

Why should a plague of this kind destroy our cattle and ruin the beef and dairy industries of the country? We were ignorant of its nature and knew not how to prevent its spread. In 1851, experiments at Pomeraye, and other places in France, made under a commission appointed by the Government, had demonstrated its infectious nature. They showed beyond question that it would spread from diseased to healthy cattle. The idea of a specific etiology had not been accepted generally. It was necessary in the circumstances, owing to popular skepticism, to repeat the experiments to convince the doubting public. This was particularly true in the United States. The American mind demanded additional evidence of its contagiousness before drastic measures for its eradication could receive approval. The work of Salmon, added to the results of the French investigators, furnished evidence of its infectiousness which made it desirable for Congress to authorize the organization of the Bureau of Animal Industry in the U. S. Department of Agriculture to carry out rigid measures for its complete eradication. As a result every trace of the disease was removed from the United States within seven years after a definite method of procedure was adopted, and at a cost ($1,609,000.72) that was trifling in comparison with the losses that would have occurred annually had it become permanently established as a plague of American cattle. It was the convincing evidence obtained from actual experimental work that made it possible to protect at that time the dairy and beef industries of the country against one of the most destructive diseases of cattle.

GLANDERS

Glanders is among the oldest equine diseases known. The ancients described it and recorded extensive losses it occasioned. Many theories prevailed concerning its cause, the most common being that it spread in some unknown way through the air. The importance of its presence is illustrated from the report of losses caused by it in Prussia where in ten years, from 1876 to 1886, it destroyed 20,566 horses. In the large cities of this country it became a veritable scourge. In 1882, Loeffler and Schutz discovered its specific organism which they were able to cultivate in pure culture. They found it escaped with the nasal discharges of infected horses and that well ones coming in contact with this material were infected. A further victory was the discovery of mallein. By its use infected animals could be detected that failed to exhibit symptoms.

Glanders, like anthrax, is communicable to man, and already a large number of human cases have been reported among those who had the care of, or employed, glandered horses. Since the introduction of mallein, the enforcement of sanitary regulations, and complete isolation or destruction of infected horses, glanders has been reduced greatly. In 1906, Robbins, of the Royal Victoria Hospital in Montreal, reported 156 cases in man. In the human species it has a very high mortality.

In the United States, glanders still prevails in certain rural districts. In other countries where automobiles are less abundant, it is a serious menace to the equine species. The horrors of strangles in countries where scientific methods for its control are not practiced, are illustrated in a report in a Soviet Red Gazette, quoted by Hobday where 117 children were destroyed because they had glanders. They broke down the sanitary requirements to prevent the disease in horses and

then shot their children when they became infected.

RABIES, OR HYDROPHOBIA

Among the diseases of man none are dreaded more, and in animals, few, if any, are more pitiable than rabies. There is no longer disagreement as to the existence and dangerous nature of this disease. It is also known that it is readily communicated from rabid dogs to healthy animals and people. With many infectious diseases the specific organism has been isolated and cultivated on artificial media. This is not true with rabies. Pasteur, by a long series of experiments, showed that the specific cause, whatever it is, was localized in the central nervous system of the diseased individual. In 1884, he demonstrated that immunity could be conferred by the use of an attenuated, fixed virus which he had been able to produce by the use of animals. Concerning his first use of the attenuated virus in man, Pasteur wrote:

> Making use of this method I had already made fifty dogs of various races and ages immune to rabies and had not met with a single failure, when on July 6th, quite unexpectedly, three persons, residents of Alsace, presented themselves at my laboratory. One of these, a boy of nine years, who had been bitten in fourteen different places by a rabid dog, was saved.

The total mortality among the first 9433 people treated was 0.161 per cent. In 1890, among 415 persons who were bitten by rabid dogs, and who took the Pasteur treatment, there was not a single death. Tens of thousands of people have taken the treatment with very few failures. In 1921, Sellers, Director of the Georgia State Board of Health Laboratories, found that rabies existed in 29 states and that a total of 5558 heads had been examined in official laboratories, and that 2690 of them were positive. Further he learned that there had been 168 cases of rabies in man from 1917 to 1921.

In 1903, Negri, of the University of Parva, described structures in the cytoplasm of brain cells of rabid animals that he suspected to be of etiological value. A few workers have arrived at the same conclusions. Others have considered these bodies to be specific degenerations. Kraus, Guerlac and Schweinberg, in their recent volume on Lyssa, place emphasis on the value of Negri bodies in the diagnosis of the disease. They are recognized as indicative of rabies in all diagnostic laboratories.

While rabies is a much dreaded disease among dogs and in the human family, it is frequently the cause of heavy losses among food-producing animals. Not long since the State Veterinarian of Wisconsin reported that in one year rabies had caused the death in that state alone of 400 cattle, 100 hogs, 56 horses and 28 sheep. In a single county in Central New York it was estimated by an official that rabies caused the death of $4,000 worth of farm animals in a single year. As rabies is transmitted from infected to healthy individuals by direct inoculation, or bites from rabid animals, and as the dog is the only animal in domestication that has a tendency to bite, the control of the disease in canines will circumscribe its ravages in other animals and in man. To accomplish this a large amount of work has been done to produce a single dose vaccine for immunizing dogs. Much progress has been made. Those engaged in the work are very optimistic that an efficient method will be forthcoming. Already the experimental work on rabies has localized its virus, shown how it is disseminated and given us effective methods for preventing the disease in people who have been exposed. These methods are equally applicable to domesticated animals, but as yet they are too expensive to be practicable.

SWINE DISEASES

The most important infectious disease of swine in the United States is hog cholera. The first recorded epizootic in

this country occurred in Ohio in 1833. It spread gradually until it became a menace to the swine industry. De Schweinitz estimated that the losses from this disease alone in one state was $15,000,000 annually. A disease supposed to have been introduced from Europe about 1830 had become a widespread and unquestioned plague by 1880. In 1903, De Schweinitz found that in the blood of swine affected with hog cholera there was a virus that would pass through a Berkefeld filter. Continued investigations in the Bureau of Animal Industry, and elsewhere, showed that swine could be immunized against this disease by the use of serum from hyperimmunized pigs. The result of this discovery has led several states to manufacture serum for the purpose of immunization. The saving from suffering and death in swine and the gain to the pork industry of the country, resulting from this work, is enormous. No one who has witnessed the loss of hogs in outbreaks of cholera can fail to appreciate the great blessing that has come to this species of animals by bringing this disease under control.

Swine plague, or *septicemia hemorrhagica*, is quite prevalent and a serum has been prepared that is reported to give excellent results in its treatment. *Swine erysipelas*, which has been a scourge of swine in certain parts of Europe, has recently been reported in this country. European observers have found human cases of infection with the organism of swine erysipelas. The symptoms are a slowly advancing, erythema-like rash, and often joint complications. Endocarditis often supervenes. Edel suggests that Rosenbach's erysipeloid is identical with the swine erysipelas as determined from the clinical, therapeutic and epidemiological aspects. Fortunately, the immunizing serum developed by Lorenz for the treatment of this disease in swine has been found to be efficient in the human subject. While at present it is rare in this country, its prevalence in Europe renders exceedingly important the production of an efficient serum.

TEXAS OR SOUTHERN CATTLE FEVER

Texas fever has been described as "bovine malaria." It has been known to exist for a great many years in the South. It frequently gained entrance to the northern states where it caused the death of many native cattle.

The cause of Texas fever was a mystery until the experiments begun in 1889 by Smith, showed it to be a protozoan, and that it was transmitted from the infected to well cattle by means of a tick (*Margaropus bovis*). It has been demonstrated repeatedly on cattle that the tick is the carrier of the virus; that in the absence of this species of tick, southern cattle can be shipped north at any time of the year with perfect safety to the native cattle; and that if the ticks are removed from the pastures in the South, northern cattle can live on them without becoming infected. These experiments led to the conclusion that the most effective manner of eliminating this destructive disease was to eradicate the cattle tick from the South. Already this has been done for large areas in the southern states with the result that the dairy industry has been improved greatly. A further and more important bearing of this discovery is that the virus of a specific disease is transmitted by an insect. This has been of great significance in the control of protozoan diseases in man that are transmitted by mosquitoes and other insects.

INFECTIOUS ABORTION IN CATTLE

For many years dairy cattle have suffered from infectious abortion which in many instances has been so extensive that the maintenance of the herd was impossible. The losses caused by it have been estimated, by the Chief of the Federal Bureau of Animal Industry, at from $20,000,000 to $30,000,000 annually. In 1897, Bang discovered its specific cause, and since that time experiments have been conducted in many countries to determine its nature and means of dissemination. Growing

out of these studies a definite method of procedure has been formulated whereby it is possible for breeders to eliminate it from their herds.

In 1918, Evans pointed out the very close relation between the cause of this disease and that of Malta fever. The similarity of the morbid conditions in goats produced by *Brucella melitensis* and that in cattle due to *Brucella abortus* is in itself suggestive that *Brucella abortus* may possess pathogenic properties for the human subject.

In 1924, Kieffer reported a case of human infection with *Brucella abortus*. The clinical picture indicated Malta fever, but the organism obtained from the blood was identified as *Brucella abortus*. DeKrote reported a case of undulant fever in a man who became infected by removing the placenta from a cow that had aborted. Duncan reports a like case in a butcher who was a heavy drinker of raw milk. Orpin found in Rhodesia an undulant fever due to the same cause. Carpenter has reported several human cases. In the last three years many reports of human infection with an organism indistinguishable from *Brucella abortus* have been made, not only in New York, but throughout the country. The investigations in connection with these cases are pointing to the fact that milk from cows that have aborted and whose udders harbor the infecting organism, may transmit it to the human subject, and that many cases of heretofore undiagnosed fever of an undulant type are caused by this infection.

Studies have been made in a large number of herds on the milk of cows that have aborted with the result that about 40 per cent of the udders of cows that actually abort harbor the organism. These experiments indicate that the safety of milk coming from herds in which this disease exists depends on pasteurization. Since July 30, 1930, all milk in California to be certified must come from cows that are free from the Bang abortion infection as well as tuberculosis. Were it not for the fact that it has been possible to use experimental animals and pregnant cattle in these investigations, the definite facts regarding the infectiousness of the milk would not have been determined.

OTHER DISEASES

In addition to the specific diseases, there are numerous parasitic infestations that cause extensive losses in all species of animals. The studies that have had for their object the ascertaining of the life history of parasites and the effect of certain drugs and chemicals on them when administered to their hosts, have made it possible, within certain limits, to control parasitism. Animal owners are indebted for their success in keeping their animals as free as they do from parasites to many long and trying investigations.

We have been dealing with the specific diseases and parasitisms. There is another field where experimentation has been productive of unmeasured benefits to animal owners. I refer to the extensive work that has been carried on in testing out the value of certain minerals in food stuffs, the effect of certain light rays on maintaining health; and the discovery of vitamins and their functions. Never before in the history of agriculture have farm animals been better nourished than at this time. Many painful disorders have been corrected through the use of proper diet. These benefits could not have come to domesticated animals had the opportunity to make long series of experiments been denied.

I have sketched in a very fragmentary manner but a part of the advantages that have come to comparative pathology and the benefits that have accrued to animal owners from the work that was started by Pasteur and his co-workers. This has stimulated greater efforts in experimental physiology from which knowledge of untold value has been acquired. In view of all that has been done, we are reminded, by some new discovery, almost daily that practical preventive medicine is in its

infancy. Many fertile fields for research are not yet entered and in none are the results final.

The demands for further investigation were never more pressing than they are today, and never before was there more interest in research. If the work should be checked, progress in animal husbandry would be doomed, and the problems associated with animal diseases would remain unsolved. The experience in this country would be like that in Great Britain, where, by parliamentary enactment, animal experimentation was so crippled that the country which should have done the most, by virtue of her wide geographic possessions and vast live stock interests, for improving methods of prevention and control of infectious diseases, has done very little. I quote from one of England's foremost veterinarians in a plea for animal experimentation:

> No country in Europe has, possibly, sustained greater loss during the last thirty-five years than our own; yet no country, perhaps, should have suffered less. With the finest breed of horses, and the most magnificent herds and flocks in the world, and a teeming population, whose health and wealth are largely centered in these, we have entirely neglected to protect them from the ravages of disease of home and foreign origin, by forgetting to foster and encourage that science which alone can accomplish this. That neglect has cost Great Britain and her colonies untold millions.[1]

[1] Fleming, G.: The Contagious Diseases of Animals; Their Influence on the Wealth and Health of the Nation, and How They are to be Combated.

BOOK REVIEWS

ARTERIOGRAPHIE DES MEMBRES ET DE L'AORTA ABDOMINALE (Arteriography of the Extremities and of the Abdominal Aorta). By Reynaldo Dos Santos, Professor to the Faculty of Medicine of Lisbon and Surgeon of the Hospitals; A. C. Lamas, Surgeon, and J. P. Caldas, Radiologist of the Hospitals of Lisbon. With a preface by Professor Leriche. Paris: Masson et Cie, 1931.

As Prof. Leriche says in the preface, here we have a beautiful book, full of original ideas, facts with a minimum of commentary, revealing new, almost undreamed of aspects of radiological investigation, a revelation likely to upset some of our present-day conceptions.

At first thought, the idea of puncturing the aorta or the larger arteries brings up visions of catastrophe, and yet, after all our work in safe use of ventriculography, in the injection of lipiodol in the spinal canal and in the tubes, is it really so revolutionary to inject into the arterial system an opaque substance which is rapidly eliminated?

The authors used abrodil or uroselectan B which is very stable and especially very soluble, thus permitting a greater opacity. Injected into the blood, it traverses the vessels as rapidly as the blood-stream, so that the radiography must be done during the injection. But the interesting story of this work is too long to relate in a review. The authors discuss fully the dangers, technique of injection, the radiographic technique. One fact deserves mention: the pain accompanying the injection of abrodil requires anesthesia—general for the upper extremities, general or spinal for the lower extremities. The authors have tried arterial anesthesia after Goyanes but not with satisfactory results. Later work with thorotrast seems to indicate it is a better medium and not painful. The radioactivity of thorotrast is so slight that no harm need be feared from that source.

The lesions in which the method has proved useful are as follows:

1. Arterial diseases: gangrene, aneurysm, ligations and resections, Volkmann's paralysis.
2. Osteomyelitis and osteoarthritis (tuberculosis and syphilis of bone, etc.).
3. Tumors of bone and of soft tissues. Of great interest are the illustrations of cases of tuberculosis of the knee and ankle joints.

For aortography the authors use uroselectan B, thus visualizing abdominal aortic aneurysms, hydatids of the liver, pelvic tumors, malarial spleen, and especially cases of nephritis, pyonephrosis and renal tuberculosis, and the demonstration of the abnormal vessels causing ureteral obstruction in certain cases of hydronephrosis. The authors' series include more than 300 cases of aortography.

HOW TO LIVE, Rules for Healthful Living Based on Modern Science. Authorized by and Prepared in Collaboration with the Hygiene Reference Board of the Life Extension Institute. By Irving Fisher, LL.D., and Eugene Fisk, M.D. Ed. 19, Completely Revised. N. Y., Funk & Wagnalls Co., 1932.

The nineteenth edition of this book, brought entirely up-to-date, is as full of meat as ever. Many physicians will undoubtedly disagree with some of the diets contained in the book but, all in all, the book is full of common sense, is practical and worth while. It is easy reading and its handsome flexible binding makes it an attractive book.

CLASSIC DESCRIPTIONS OF DISEASE. By Ralph H. Major, M.D., Springfield, Ill., Charles C. Thomas Co., 1932.

Over 300 selections of original general discussion with 127 illustrations and biographical sketches of the authors making an interesting anthology. Neither the reviewer nor anyone else would have made exactly the same selections. It is easy to criticize any anthology on the basis of both inclusion and exclusion. However, it must be admitted that the author has done his job well and little, if any, objection can be taken to the inclusion of the splendid pages he has selected from the works of the great medical authors from Hippocrates through Mackenzie. His biographies are concise, well written and informative. The selections included are entirely from the realm of internal diseases and it would be interesting to have a similar volume on surgery.

A Descriptive Atlas of Radiographs, An Aid to Modern Clinical Methods. By A. P. Bertwistle, M.B., CH.B., F.R.C.S. Ed. 2, Revised and Enlarged, St. Louis, C. V. Mosby Co., 1932.

This is the second edition of this work with 767 radiographs collected from various sources. The leading English physicians have contributed to the volume. The text consists of descriptions of the radiographs with short chapters on Radio-Diagnosis, The Clinical Application of Radiology, and Milestones in Radio-Diagnosis. This book may be a handy work of reference for the tyro but we believe the more experienced man may find it necessary to have more complete monographs covering the various chapters.

The subjects covered are: Normal Bones and Epiphyses, Congenital Abnormalities, Fractures, Inflammation of Bones, Tumours of Bone, Injuries and Diseases of Joints, Nasal System, Alimentary System, Urinary System, Respiratory System, Nervous System, Vascular System, Thyroid System, Female Generative System and Muscles. In attempting to cover such a field in a volume of 550 pages, many loopholes are to be found.

As far as it goes, the work has been well done but it is felt that it is too sketchy to be recommended as a practical reference work.

Fungous Diseases, A Clinico-Mycological Text. By Harry P. Jacobson, M.D., With Introductions by Jay Frank Schamberg, M.D. and Howard Morrow, M.D. Springfield, Ill., Charles C. Thomas Co., 1932.

As Dr. Schamberg points out in his Introduction, "While there is still much to be learned with regard to many points in connection with the life-history of the diseases described, a great mass of clinical and laboratory facts have already been garnered, and these are most skillfully presented by Dr. Jacobson." The book lives up to its title and will be found a valuable monograph for reference on the subject treated. The illustrations are splendid and the bibliography seems to be complete. Dermatologists and pathologists should find this book of considerable interest.

Individuality of the Blood in Biology and in Clinical and Forensic Medicine. By Prof. Leone Lattes, Translated by L. W. Howard Bertie, M.A., B.M., B.CH. (Oxon.) From the French Edition of 1929, Thoroughly Revised and Brought Up to Date by the Author. Oxford Univ. Press, 1932.

This book, originally published in Italy in 1923, has since been translated into German and French and, in its English translation, will undoubtedly be of value to a number of readers. The bibliography of almost 100 pages is in itself evidence of the thoroughness of the author's work. While not all will agree with the author's theories, all hematologists will want to have the book at hand for study and reference.

Livre d'or à l'occasion du jubilé de vingt-cinq ans d'activité chirurgicale du Docteur Theodore L. Papayoannou, Ancien Professeur d'Anatomie à l'Ecole des Beaux-Arts d'Athènes, Directeur et Chirurgien en Chef de l'Hôpital Papayoannou. Naumburg-Saale, Lippert & Co., 1932.

This is an interesting anniversary volume published in honor of the Greek surgeon's golden anniversary in surgery. Articles are in Greek, German and French.

Kosmetische Operationen, Ein kurzer Leitfaden für den Praktiker. By Dr. Ernst Eitner. Wien, Julius Springer, 1932.

This little monograph in 130 pages covers in sketchy and concise fashion the entire subject of cosmetic operations and will serve as a résumé of the possibilities of cosmetic surgery. It is well illustrated and the bibliography makes it a simple matter to look up further references.

Handbuch der gesamten Unfallheilkunde. By Drs. Fritz Konig and Georg Magnus. Stuttgart, Ferdinand Enke, 1932.

The latest part of this work begins Volume II dealing with Industrial Diseases. In this volume, the relation of Industry to Diseases like Lead Poisoning, etc. is taken up in thorough detail. We look forward to a completion of this work which we believe to be one of the most valuable reference textbooks for the industrial surgeon.

Mesenteric Lymphadenitis, A Clinical Study. By J. P. Strombeck. Stockholm

Kungl. Boktryckeriet, P. A. Norstedt & Sons, 1932.

Strombeck, working in Soderlund's clinic in Stockholm, offers in this monograph a careful study of a large series of patients showing by radiographic examination calcification of their mesenteric lymph nodes. The work includes an extensive review of the literature of mesenteric lymphadenitis; also a report of a limited number of cases of acute lymphadenopathies. The material has been carefully analyzed. Unfortunately the results of this analysis, in terms of differential diagnosis, are sufficiently equivocal to leave the reader with but hazy ideas as to how this study may be applied with specific cases. The limited amount of pathological study of the non-calcified nodes leaves many of the conclusions open to question. While the work, for example, represents a clinical study of mesenteric lymphadenitis no mention is made of Hodgkin's disease which, in reality, affects the mesenteric nodes more than any other group within the body. Tabes mesenterica, likewise, receives no emphasis. The monograph must be viewed, therefor, as almost exclusively a study of *calcified* nodes rather than as a broad exposition on intra-abdominal lymphadenopathies in general. The section reviewing the extensive literature proves the portion of perhaps greatest merit. The illustrations are clear and the book is well edited. It should prove of great interest not only to the student of tuberculosis but to the clinician intent upon solving the problem case with abdominal signs and symptoms.

Endocrine Medicine. By William Engelbach, M.D., F.A.C.P., B.S., M.S., D.SC. With a Foreword by Lewellys F. Barker, Springfield, Ill., Charles C. Thomas, 1932. 3 Vols. and an Index Vol. Vol. I. General Considerations; Vol. II. The Infantile Endocrinopathies, The Juvenile Endocrinopathies; Vol. III. The Adolescent Endocrinopathies, The Adult Endocrinopathies.

As Dr. Barker well states in his foreword: "It is a matter of congratulation, that Dr. William Engelbach should have been willing, in the light of his large personal clinical experience, and after a painstaking study of the older and newer literature, to provide us with a summary of endocrinological theory and practice as known in 1931."

This work is published in three volumes of over 1800 pages to be followed by an index volume, and containing 933 illustrations. As a book of reference for the close student who wishes his data complete and up-to-date, there is nothing better in the English language today. The subject is completely covered and well handled though perhaps the average practitioner will find himself lost in the mass of detail. The subject, after all, is new and ideas are changing. No work, therefore, however well done or authoritative, is to be considered as the final word, for changes take place in this subject from day to day. This set will undoubtedly take its place as a definite landmark in the history of endocrine literature and will probably stand as the authoritative presentation of the subject to the end of 1931.

It is because of the subject treated rather than any fault of the book itself that it will before very long need to be revised. We look for much more research and many more publications before a standardization of this subject can possibly be achieved. In the meantime, the author and the publisher are to be congratulated on the most complete presentation of the subject to date.

Diseases of the Spinal Cord. By Williams B. Cadwalader, M.D. Introduction by William G. Spiller, M.D. Balt., Williams & Wilkins Co., 1932.

This book of 200 pages discusses the diseases of the spinal cord in a rather elementary manner. It would seem to be more suitable as a section of a book on nervous diseases than as a separate monograph. The work is based on the lectures given by the author to his students at the University of Pennsylvania. Desirable as it might be to do so, it is doubtful whether many teachers will feel justified in recommending to their students a special monograph on diseases of the spinal cord. The book is well written, well printed, and well illustrated.

The Principles of Orthopedic Surgery for Nurses. By James Warren Sever, M.D. Ed. 2, N. Y., Macmillan Co., 1932.

The second edition of Sever's book brings the subject thoroughly up-to-date in a concise and practical manner. All the information needed by nurses is very apt to be found in this volume and Dr. Sever has avoided the temptation, so common in many of the books on nursing, of overdoing the subject. He has told the nurse all she needs to know, no more, no less, and is to be congratulated on his accomplishment.

PERIPHERAL NERVE INJURIES

LEWIS J. POLLOCK, M.D., AND LOYAL DAVIS, M.D.

ELEVENTH INSTALLMENT

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Chapter XXXII

THE TIBIAL NERVE

In lesions above the popliteal space a complete isolated injury to the tibial nerve is rare. More frequently, as was noted under lesions of the sciatic nerve, partial or dissociated paralysis of the nerve accompanied by more or less complete paralysis of the peroneal nerve occurs. In lesions below the popliteal space the tibial may be completely severed.

Motor Symptoms

The most common motor disturbance which results from a lesion of the tibial nerve is a paralysis of plantar flexion of the foot. Frank adduction of the foot is also impossible. It is always accompanied by elevation of the foot and is then due to contraction of the tibialis anticus. Flexion and separation of the toes are abolished and no muscle or tendon can move in the sole.

The foot may be plantar flexed by the action of the peroneus longus, but feebly. Walking is fatiguing, difficult and often painful. The foot hangs down, is swollen, edematous and discolored. The proximal phalanges are extended, the distal ones flexed. Hyperkeratosis of the sole and ulceration are common. Often the foot is deformed by fibrosis of the tibiotarsal articulation, and the attitude of foot-drop cannot be corrected passively. At times dorsal flexion of the proximal phalanges may produce passive flexion of the distal phalanges and claw-foot result (Fig. 282).

Sensory Symptoms

Subjective sensory disturbances, frequently of a causalgic character, are very common in injuries of the tibial nerve, particularly in incomplete lesions (Fig. 283).

Objective sensibility is lost over the sole, except at the inner border, the lateral surface of the heel, the plantar sur-

face of the toes and at times encroaches on the distal phalanges. The area of the loss of sensation may be seen in the preceding figure. The area of overlap of adjacent nerves includes the

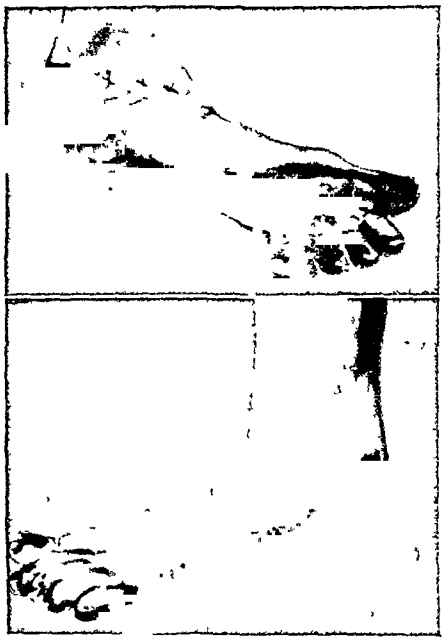


FIG. 282. Paralysis of tibial nerve.

inner border of the sole, much of the lateral surface of the heel and in some cases we have found that it included the plantar surface of the areas between the toes (Fib. 284).

The area of total supply for pain of the tibial nerve was obtained from the residual sensibility in a case of a combined lesion of the small sciatic, the long saphenous, and the peroneal nerves from a case of peroneal section. This is shown in Figure 285.

VASOMOTOR AND TROPHIC SYMPTOMS

The vasomotor and trophic changes are much the same as those seen in sciatic nerve lesions. The symptoms include discoloration of the skin, which may be livid, purplish and glossy; coldness, trophic ulcers of the malleoli, the heel and toes; nail changes; hypotrichosis and hypertrichosis; and edema.

Lesions of the Posterior Tibial Nerve

Paralysis of the posterior tibial nerve frequently occurs as the result of injury in the calf, and produces a paralysis of all

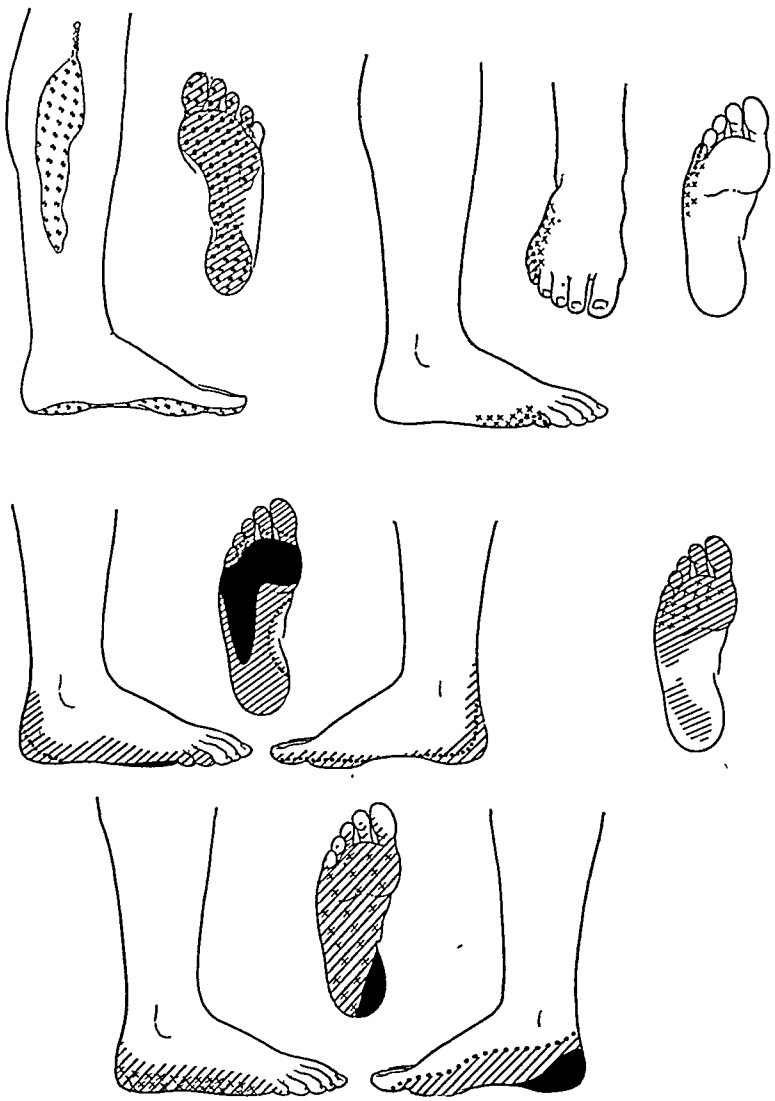


Fig. 283. Sensory loss in partial lesions of tibial nerve.

the muscles of the sole. The nerve lesion very frequently is accompanied by pain of a causalgic nature. At times a dis-

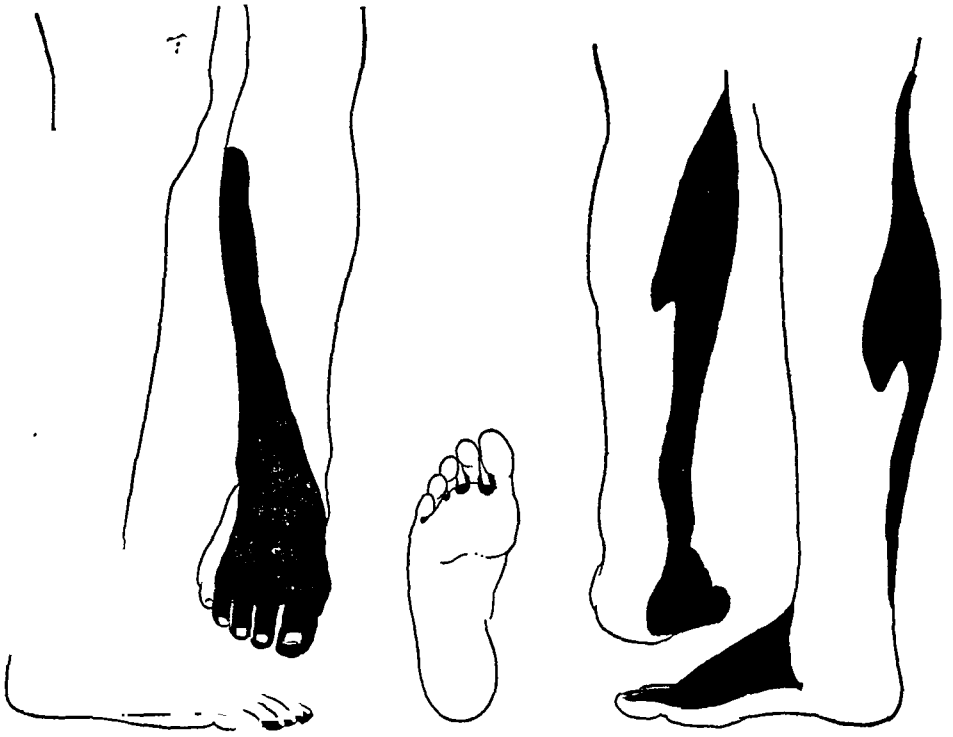


FIG. 284. Overlap of peroneal into tibial nerve area for pain sensation.



FIG. 285. Residual sensibility in tibial nerve lesions.

sociated paralysis of the plantar muscles may be found. Contractures are common, much as they are in ulnar nerve lesions, with dorsal flexion of the proximal and plantar flexion of the



FIG. 286. Paralysis of posterior tibial nerve.

distal phalanges (Fig. 286). In addition to dissociated paralyses, many of these cases showed only partial injury to parts of the posterior tibial or tibial nerves. The sensory disturbances may be visualized by reference to the preceding figure.

ANATOMY

The tibial is the larger of the two terminal divisions of the sciatic trunk and as has been stated arises from the anterior branches of the last two lumbar and first three sacral nerves. It occupies the internal portion of the sciatic trunk in its course through the thigh. After it separates from the peroneal, the tibial descends through the middle of the popliteal space to the lower part of the popliteus muscle. Here it passes beneath the arch of the soleus muscle accompanied by the popliteal artery and terminates as the *posterior tibial* nerve. It is overlapped by the hamstring muscles above and then becomes quite superficial. Opposite the knee joint it is in close relation to the popliteal vessels lying upon the popliteal vein with the artery below the vein. Below this it is again overlapped by the gastrocnemius muscle as it passes between its two heads (Fig. 287A).

The branches of the tibial nerve are *articular, muscular* and one *cutaneous* branch. The articular branches are three in number and supply the knee joint. They are difficult to dissect

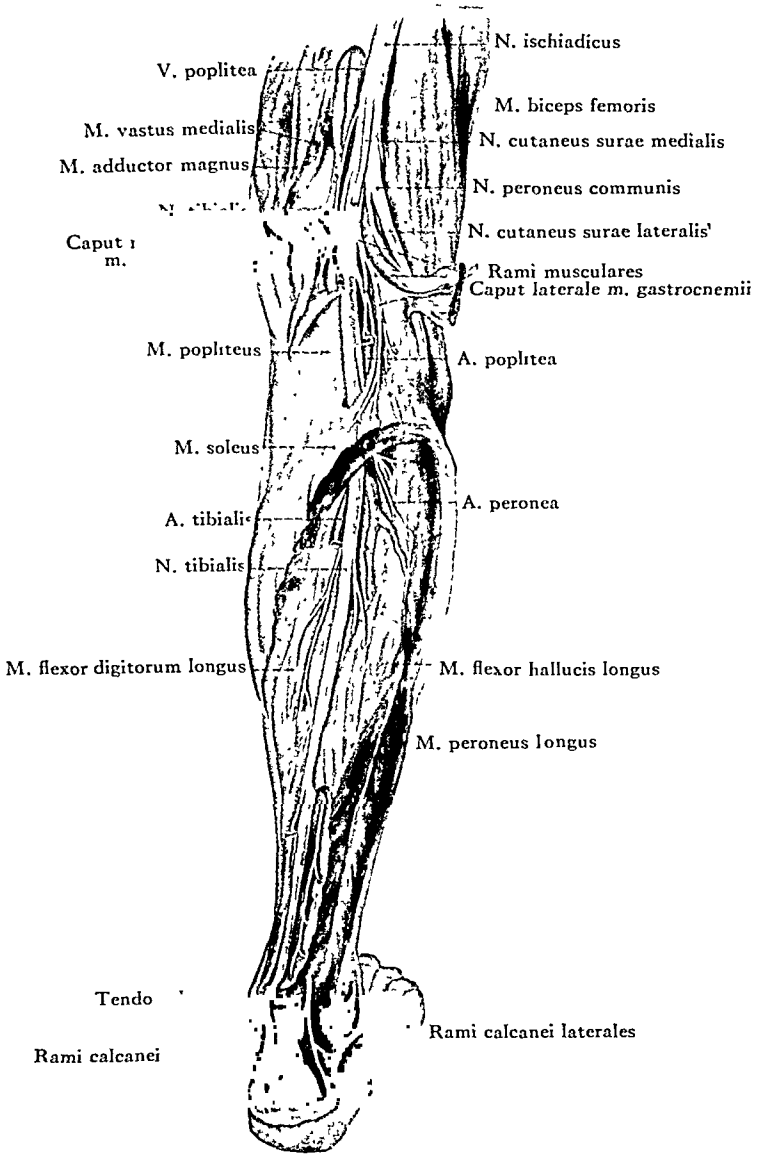


FIG. 287A. Anatomical course of tibial nerve.

but two of them accompany the superior and inferior internal articular arteries while the third follows the azygos articular artery. Four or five muscular branches are given off in the

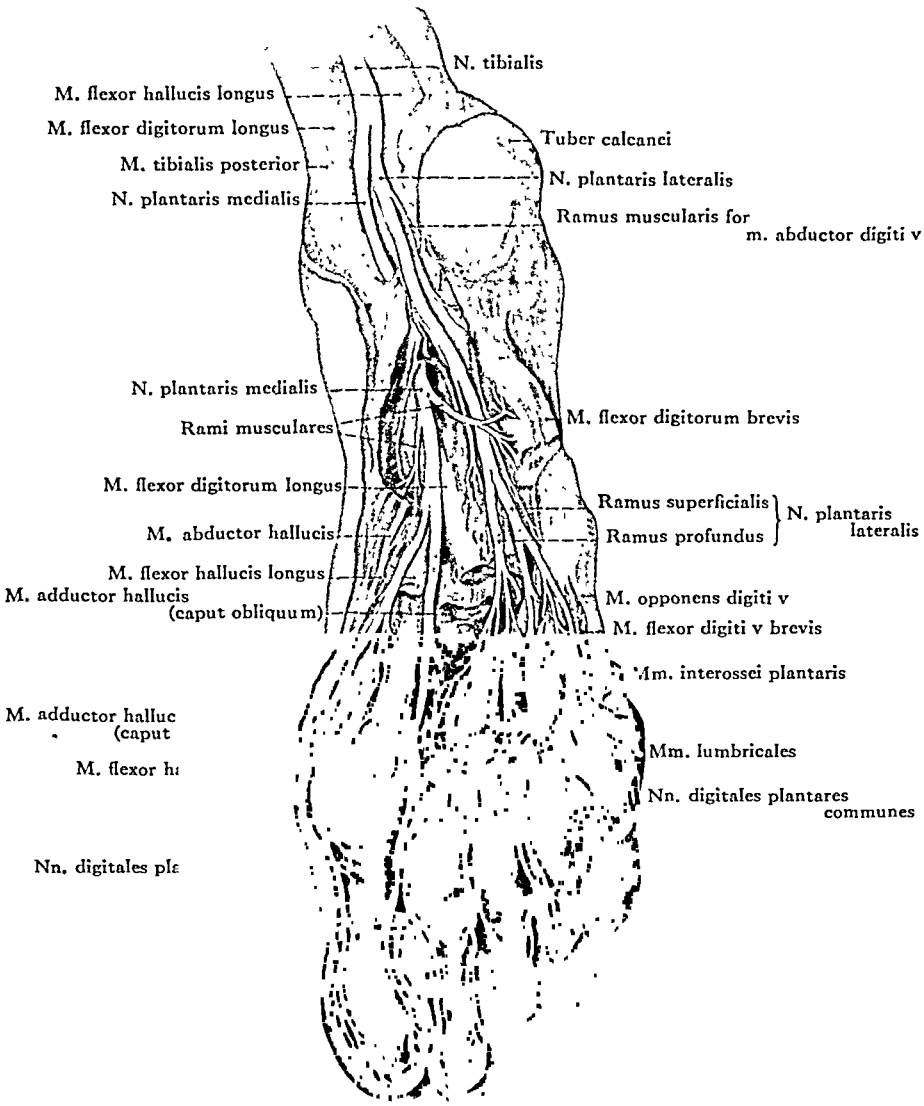


FIG. 287B. Terminal branches of tibial nerve.

popliteal space. Two of these arise from the tibial nerve near the apex of the popliteal space. They diverge from the nerve

trunk and enter the medial and lateral heads of the gastrocnemius muscle. A branch to the soleus muscle may arise from the tibial trunk or from the branch to the gastrocnemius. It

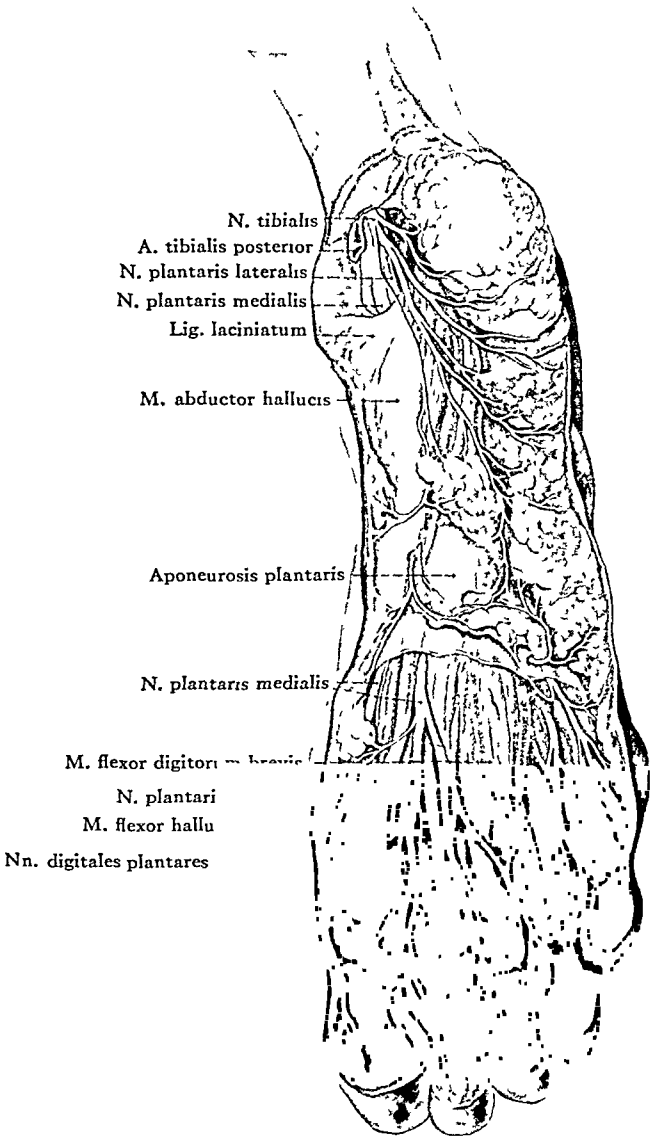


FIG. 287C. Superficial plantar nerves.

enters the superficial surface of the soleus just in front of the lateral head of the gastrocnemius. The plantaris and the

popliteus muscles are also supplied by branches of the tibial given off at this level. The *medial sural cutaneous nerve* (communicans tibialis) descends between the two heads of the gastrocnemius and pierces the deep fascia about the middle of the posterior surface of the leg. It joins a branch from the peroneal to form the external saphenous nerve to the interval between the os calcis and external malleolus. Here it divides into an anterior branch which supplies the skin along the outer side of the foot and little toes; and a posterior branch which forms the lateral calcaneal cutaneous nerve.

The *posterior tibial* nerve, which is the continuation of the tibial trunk, descends along the back part of the leg with the posterior tibial vessels to the interval between the internal malleolus and the os calcis. Here it divides into its terminals, the *internal* and *external plantar* nerves. The nerve is covered by the muscles of the calf and at a lower level only by the skin and fascia. Above it lies to the inner side of the tibial artery but it crosses this vessel and then lies externally as far as the ankle. In the lower third of the leg it is parallel to the Achilles tendon.

The collateral branches of the posterior tibial nerve are the *articular* which supplies the ankle joint; the *internal calcaneal* which supplies the skin of the heel and inner side of the foot and the *muscular* branches. The latter supply the deep surface of the soleus, posterior tibial, flexor longus digitorum and the flexor longus hallucis muscles. Thus the soleus receives a double nerve supply, one deep and one superficial.

The *internal plantar*, the larger of the two terminal branches of the posterior tibial, passes forward beneath the abductor hallucis muscle and between it and the flexor brevis digitorum to divide opposite the metatarsal-tarsal articulations into four *plantar digital* nerves. During its course, it gives off cutaneous branches which supply the skin of the sole of the foot and muscular branches which supply the abductor hallucis and flexor brevis digitorum muscles. Articular branches supply the tarsal-metatarsal articulations. The four terminal

digital branches are distributed similar to the digital branches of the median nerve in the hand. The first supplies the skin over the inner border of the large toe and innervates the flexor brevis hallucis muscle. The second supplies the skin of the adjacent sides of the large and second toes and innervates the first lumbrical muscle. The third supplies the cutaneous innervation for the adjacent sides of the second and third toes, and the fourth supplies the skin over the corresponding sides of the third and fourth toes. (Fig. 287B and C.)

The *external plantar*, which is the smaller terminal of the posterior tibial, passes forward with the external plantar artery to the outer side of the foot. It lies between the flexor brevis digitorum and flexor accessorius.

In the interval between the former and the abductor minimi digiti, it divides into a superficial and deep branch. Before this division occurs it supplies the flexor accessorius and the abductor muscles. The superficial branch separates into two digital nerves, one of which supplies the outer side of the little toe, the flexor brevis minimi digiti and the two interosseous muscles of the fourth metatarsal space. The other supplies the adjacent sides of the fourth and fifth toes. The deep branch accompanies the external plantar artery beneath the tendons of the flexor muscles and the adductor obliquus hallucis muscles. It supplies all the interossei, with the exception of those in the fourth metatarsal space, the three lateral lumbricales, the adductor obliquus hallucis and the adductor transversus hallucis muscles. It will be recognized that the distribution of the external plantar is similar to that of the ulnar.

PHYSIOLOGY

The *popliteus* muscle flexes the leg upon the thigh.

The *plantaris* is inconstant and has no specific action.

The *gastrocnemius* and *soleus* have a united action. They plantar-flex the foot in adduction.

The *tibialis posticus* muscle is a pure adductor of the foot.

The *flexor longus digitorum* flexes the terminal phalanges of the four smaller toes.

The *flexor hallucis longus* flexes the second phalanx of the great toe.

There are three muscles in the foot which correspond to the muscles of the thenar eminence in the hand. The *adductor obliquus hallucis* separates the large and second toes. The *flexor brevis hallucis* flexes the first phalanx of the large toe on the metatarsal bone. The *abductor hallucis* flexes the large toe and brings it nearer the second toe.

There are three muscles which correspond to the hypothenar eminence. These are the *abductor minimi digiti*, the *flexor brevis minimi digiti* and the *opponens minimi digiti*. All of them flex the small toe upon the fifth metatarsal bone.

In addition the *flexor brevis digitorum* muscle flexes the second phalanx of the four smaller toes.

There are *lumbrical* muscles similar to those in the hand and three plantar and four dorsal interossei muscles. They flex the first and extend the last phalanges of each toe. The interossei also abduct and adduct the toes.

Surgery

The incision used for exposure of the popliteal space may be continued down the leg to expose the tibial nerve in the upper part of its course. It lies between the intermuscular septum between the deep and superficial muscles of the calf (Fig. 288). It passes through the tendinous arch of the soleus muscle with the tibial muscles. The gastrocnemius should be retracted and the union between its two heads separated. If it is desired to expose the nerve in the middle or lower portion of the leg, an incision should be made along the medial border of the calf about 3 to 4 cm. from the margin of the tibia. The deep fascia, with its transverse fibers, is cut and the intermuscular septum identified. The nerve in this

position lies lateral to the artery. The lower in the leg the tibial nerve is sought the easier becomes its exposure.

A curved incision about 10 cm. long parallel to the dorsal

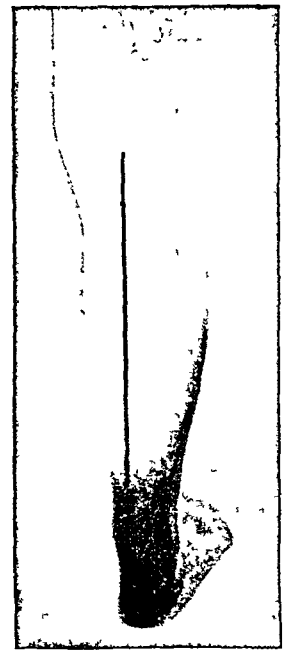


FIG. 288. Line of incision to expose tibial nerve.

border of the medial malleolus should be made to expose the internal and external nerves. The internal arcuate ligament should be exposed and divided. Immediately behind the malleolus are the sheaths for the tibialis posterior and flexor digitorum longus tendons. Below the latter are the plantar nerves and vessels. They enter the sole of the foot lying on the deep surface of the abductor hallucis muscle.

Chapter XXXIII

THE LUMBOSACRAL PLEXUS

Intrapelvic injury to the lumbosacral plexus may result from fractures and dislocations of and about the pelvis, from gunshot and stab wounds and from compression. Such isolated paralyses have only rarely been observed. In such a case Hartman reported a paralysis of the sciatic, obturator, femoral and gluteal nerves. Isolated paralysis of the ilio-hypogastric nerve with paralysis of a segment of the abdominal wall and with loss of sensation over the lateral lower abdomen has been observed. Paralysis of the obturator with paralysis of all of the abductors of the thigh and no sensory loss has likewise been observed. A lesion of the genitofemoral nerve has been noted by Leri and Bouvent. In this case sharp pain in the inguinal region at the right side of the scrotum radiated to the thigh. There was retention of urine, followed by painful micturition and anesthesia on the upper part of the thigh and external iliac fossa.

The *pudic nerve*, when found injured on rare occasions, gave rise to painful sensations. There was difficulty and burning on micturition and defecation; partial incontinence of urine and feces; pain in the anus, perineum and one side of the penis; and a semi-erection on micturition.

Lesions of the *lateral cutaneous nerve*, so often involved in civil life in neuralgia and paresthesia, gave rise to neuralgic pains similar to those seen in causalgia. Others showed nothing but sensory loss and required no treatment.

The *small sciatic nerve* was injured frequently in conjunction with lesions of the sciatic and produced loss of sensibility over the posterior surface of the thigh (Fig. 289). At times hyperesthesia was noted.

The important branches of this plexus which require description are the gluteal nerves, the femoral, the sciatic and its branches.

GLUTEAL NERVES: The *gluteal nerves* are rarely injured alone. The inferior gluteal, which supplies the gluteus maximus, was injured more frequently than the superior. The gluteus

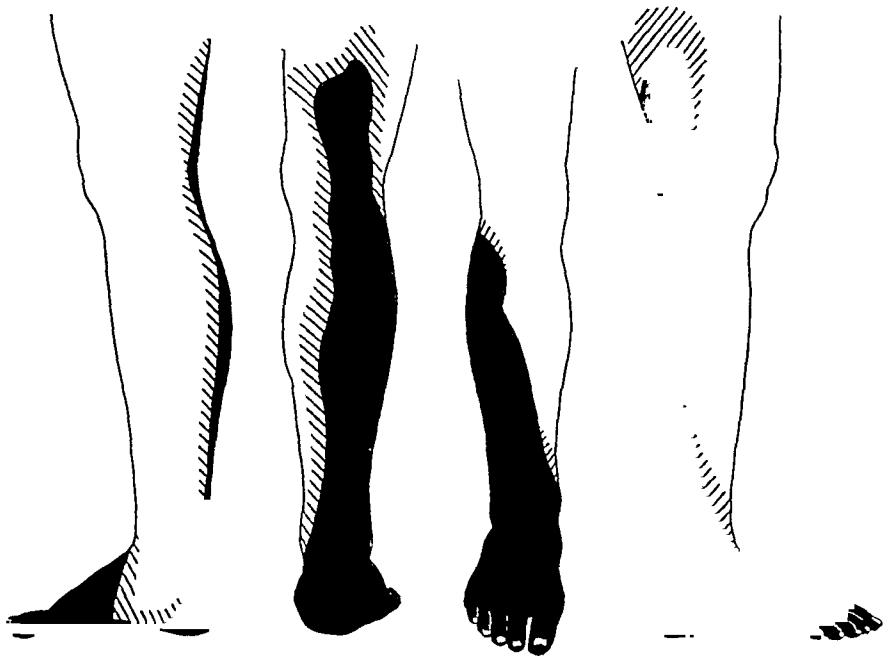


FIG. 289. Sensory loss in a lesion of small sciatic nerve in addition to a lesion of sciatic nerve.

maximus is a very powerful extensor of the thigh on the pelvis. When it is paralyzed, there is difficulty in raising oneself from a seated position, and difficulty is experienced in going upstairs, jumping and running. When unilaterally injured, the paralysis may be determined by having the patient lie upon the abdomen and by contraction bring the buttocks together (Fig. 290).

The superior gluteal nerve supplies the gluteus medius and minimus. Their action produces adduction of the thighs. It plays an important part in the fixation of the pelvis when the hip is flexed, as in the second phase of walking. When paralyzed, there is an inclination of the pelvis to the opposite side when the patient stands on the affected limb.

In testing these muscles the patient is instructed to resist passive adduction of the abducted thigh.

The rarity of observations upon isolated injuries to the intrapelvic branches of the lumbosacral plexus is due largely to the serious character of the wounds which produce them.



FIG. 290. Paresis of left gluteal muscle due to stab wound nerve lesion.

The lumbosacral plexus may be more easily understood if it is considered in its two components; the lumbar plexus and the sacral plexus. However, it is of simple structure as compared to the brachial plexus.

ANATOMY OF THE LUMBAR PLEXUS

The lumbar plexus consists of a union of the anterior branches of the first three lumbar spinal nerves. Occasionally

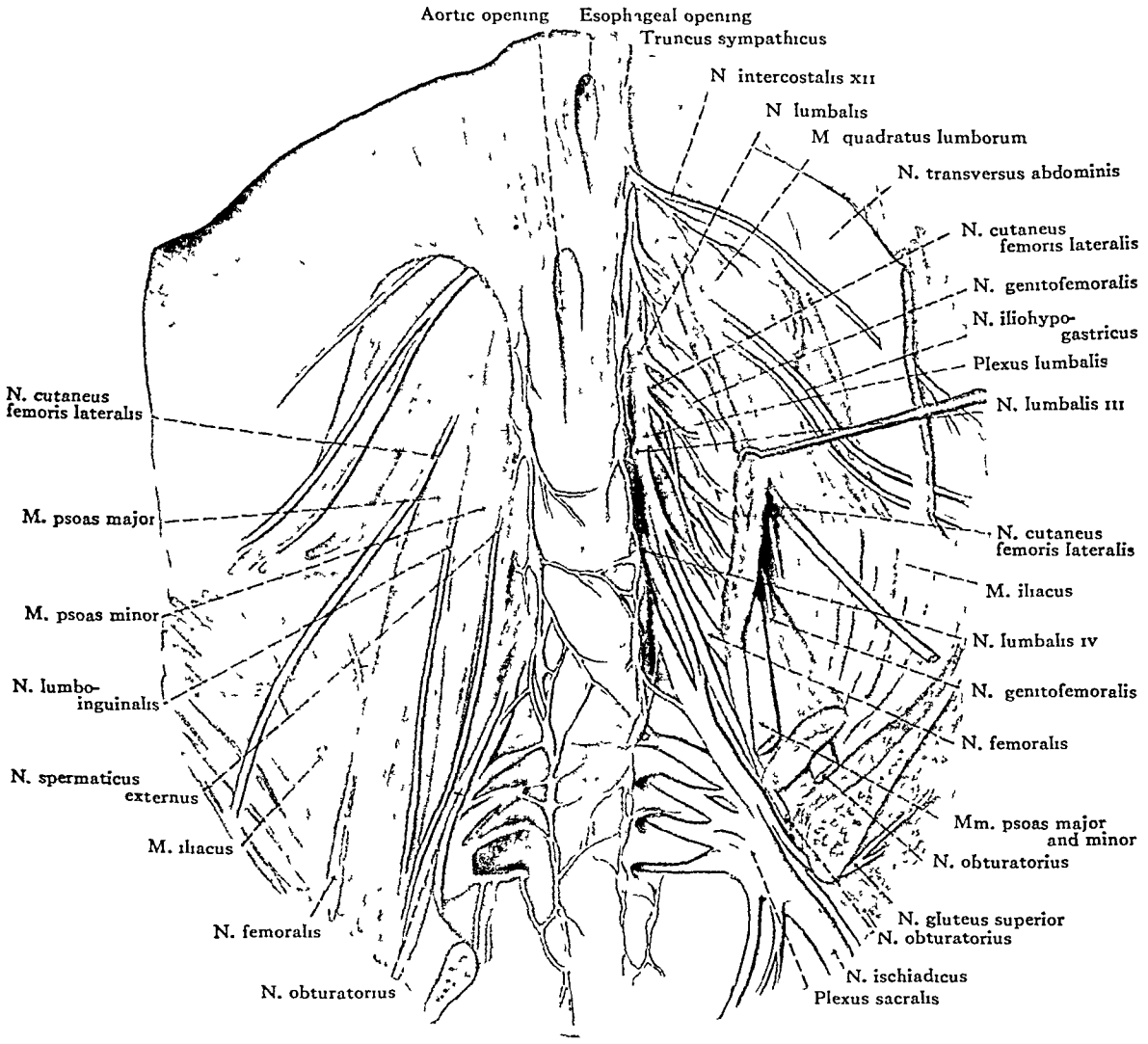


FIG. 291. Diagram of lumbosacral plexus.

it receives a contribution from the fourth lumbar and more often one from the twelfth dorsal. It is situated within the substance of the psoas muscle (Fig. 291).

The anterior branch of the first lumbar nerve receives an anastomosing twig from the twelfth spinal nerve and then divides into three branches. Two of these pass forward parallel to the twelfth intercostal and become the iliohypogastric and the ilioinguinal nerves. The remaining branch descends along the vertebral column and unites with the second lumbar nerve.

The anterior branch of the second lumbar nerve also gives off three branches. One of these passes forward and becomes the genitofemoral nerve; another passes more posteriorly and becomes the lateral cutaneous nerve of the thigh while the third branch which lies between these two descends vertically and after bifurcating sends one twig to the femoral and another to the obturator nerves.

The anterior branch of the third lumbar nerve quickly divides into a larger division which becomes the superior root of the femoral nerve and a smaller division which becomes the superior root of the obturator nerve.

The anterior branch of the fourth lumbar nerve almost immediately divides into three branches. The superior of these unites with the femoral nerve, the middle with the obturator and the inferior descends to join the fifth lumbar spinal nerve. The femoral and obturator nerves, therefore, are the terminal branches of the lumbar plexus.

All of the roots of the lumbar plexus are not situated upon the same plane. The superior and inferior roots are more anteriorly placed than the middle roots due to the normal curvature of the lumbar vertebral column. The plexus is situated in a plane of cleavage between two layers of the psoas muscle. There the nerves are in relation with the lumbar veins and arteries. The veins may pass either anterior or posterior to the nerves and occasionally they may divide to enclose the nerves in a loop. The intercostal branches of the lumbar arteries pass anterior to the nerves of the plexus. (Fig. 292.)

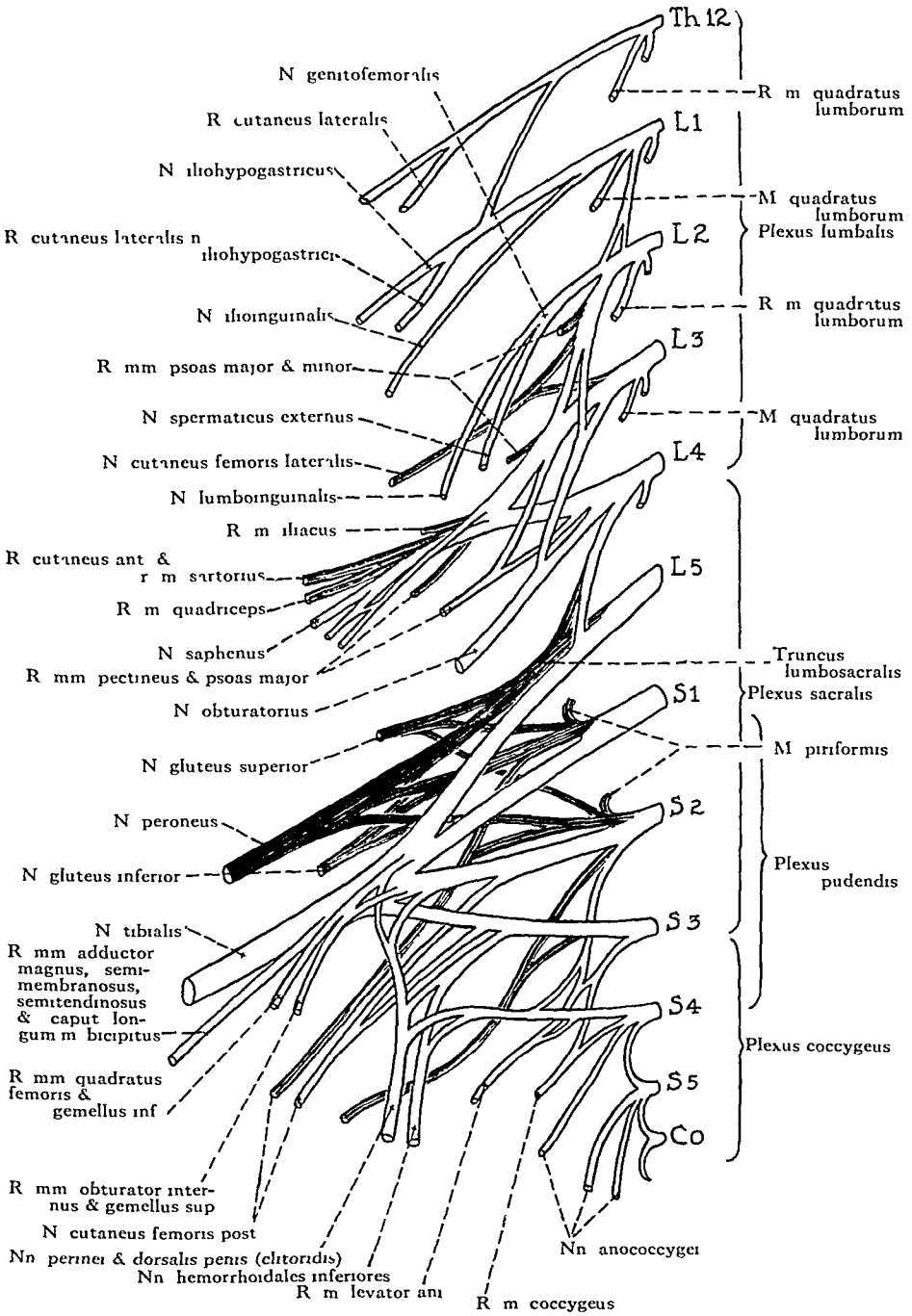


FIG. 292. Drawing to illustrate anatomy of lumbar plexus.

The white and grey rami communicantes between the nerves of the lumbar plexus and the sympathetic system are very irregular in their disposition. There may be only one pair of rami communicantes or there may be three and Hartman has described five rami connected with the first lumbar nerves alone.

A small short twig arises from each of the anterior branches of the first four lumbar spinal nerves near their origin and supplies the four lumbar intertransversarius muscles.

The *nerves to the quadratus lumborum* muscle arise from the first three lumbar nerves immediately lateral to the above named branches. They pass behind the plexus and penetrate the posterior layer of the psoas after which they pierce the anterior surface of the quadratus lumborum.

The *nerves to the psoas* muscle are variable in number although it is more common for there to be three or four. They arise from the superior root of the femoral nerve and from the common trunk formed by the middle roots of both the femoral and obturator nerves. The nerves course inferiorly and externally in front of the plexus and ramify extensively before penetrating the posterior surface of the anterior layer of the psoas muscle.

The *iliohypogastric* nerve arises from the anterior branch of the first lumbar nerve. It passes freely and externally within the substance of the psoas muscle parallel to and between the twelfth and intercostal and ilioinguinal nerves. It appears at the external border of the psoas and lies upon the quadratus lumborum. The nerve then passes still more obliquely inferiorly and laterally and at the external border of the quadratus it perforates the transversalis fascia just above the crest of the ilium. It gives off muscular branches to the abdominal wall and divides into its terminal iliac and hypogastric branches. The former perforates the internal and external oblique muscles and supplies the skin of the gluteal region. The latter runs forward between the internal oblique and transversalis muscles. It pierces the internal oblique and becomes cutaneous

by perforating the external oblique just above and lateral to the external abdominal ring. It supplies the skin over the hypogastric region.

The *ilioinguinal* nerve arises from the anterior branch of the first lumbar nerve. It appears at the lateral border of the psoas just below the iliohypogastric. It passes obliquely across the quadratus lumborum and pierces the transversalis muscle near the crest of the ilium. It communicates with the iliohypogastric between the transversalis and internal oblique muscles. It then pierces the internal oblique as it passes forward and medially and gives off branches to that muscle. The nerve accompanies the spermatic cord through the external abdominal ring. It becomes superficial and supplies the skin of the upper and inner part of the thigh, the root of the penis and the scrotum in the male and to the skin covering the mons veneris and labium major in the female.

The *genitofemoral* nerve arises from the anterior branch of the second lumbar nerve but it is not uncommon for it to have an accessory root from the first lumbar. It passes obliquely through the psoas muscle and appears at its medial border at the level of the third lumbar intervertebral space. It descends on the anterior surface of this muscle, lying behind the peritoneum, and at a variable level divides into two terminations. The genital or external spermatic branch pierces the transversalis fascia, or passes through the internal abdominal ring. In the male it follows along the posterior surface of the spermatic cord into the scrotum and supplies the cremaster muscle. In the female it accompanies and supplies the round ligament. The femoral branch descends with the external iliac artery beneath Poupart's ligament and over the sartorius muscle. It divides into an anterior and posterior branch which supply the skin of the anterolateral and posterolateral surfaces of the thigh. The terminal branches of the lumbar plexus are the femoral and obturator nerves. They will be considered separately in more detail.

Physiology of the Lumbar Plexus

The *quadratus lumborum* muscle draws down the last rib. If the thorax and vertebral column are fixed the muscles acting together may flex the trunk.

The iliopsoas muscle flexes the thigh on the pelvis and with the femur fixed both of these muscles bend the lumbar portion of the vertebral column and pelvis forward.

Surgery of the Lumbar Plexus

Because of their comparatively long course within the spinal canal the nerves of the lumbar plexus are involved more commonly in lesions affecting the lower thoracic and upper lumbar spinal column. In civil life these lesions include tumors of the cauda equina, fracture dislocations of the vertebrae, and bullet injuries of the spinal canal. The surgical approach to the cauda equina is, of course, by a laminectomy of the lumbar vertebrae. If divided within the canal, the nerves may be sutured since their structure is that of a peripheral nerve though they lie within the dural sac. The nerves are less commonly injured in their course outside the spinal canal and when they are so involved there is usually a large destructive lesion of the soft tissue which overshadows the nerve lesion. Attempts to isolate and suture the nerves in their peripheral course are impractical.

Anatomy of the Sacral Plexus

The sacral plexus is formed by the lumbosacral cord and the anterior branches of the first sacral and portions of the second and third sacral spinal nerves. The lumbosacral cord consists of the union of a part of the anterior branch of the fourth lumbar and the entire anterior branch of the fifth lumbar nerves (Fig. 293).

The plexus lies against the posterior wall of the pelvic cavity and rests upon the anterior surface of the pyriformis muscle. It has the shape of a right-angled triangle, two sides

of which are formed by the anterior branch of the third sacral nerve and the vertebral column. The hypothenuse of the triangle is formed by the lumbosacral cord and the apex is at

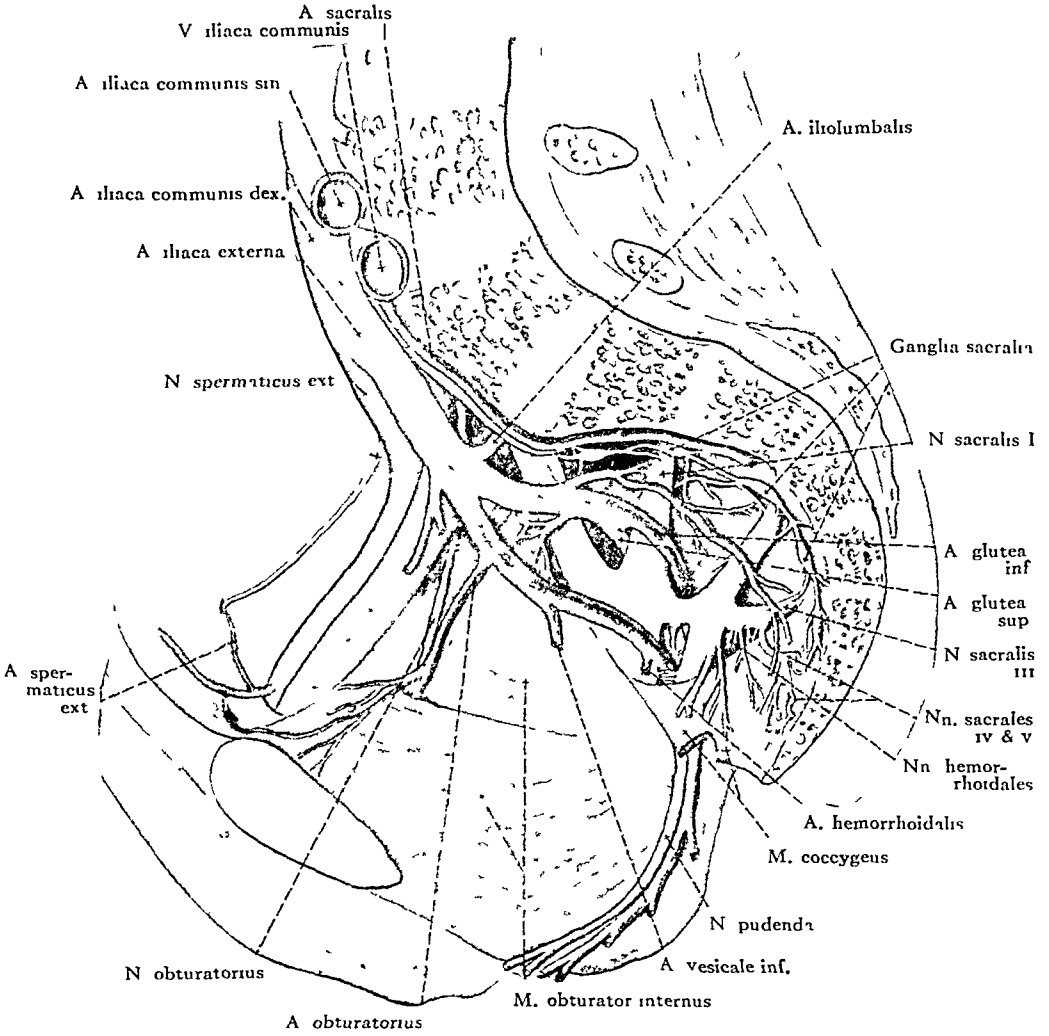


FIG. 293. Drawing to illustrate anatomy of sacral plexus.

the lower part of the greater sacrosciatic foramen. It is covered anteriorly by the pelvic fascia which separates it from the internal iliac vessels, the ureter and the pelvic colon. The gluteal blood vessels run between the lumbosacral cord and

the first sacral nerve, and the sciatic vessels between the second and third sacral nerves.

Each of the spinal nerves which contribute to the formation of the plexus receives a grey ramus communicans from the sympathetic ganglia. A white ramus from the third sacral and occasionally from the second and fourth sacral nerves is given to the sympathetic pelvic plexus.

The nerve to the quadratus femoris and gemellus inferior; the nerve to the obturator internus; the nerve to the pyriformis; the superior and inferior gluteal nerves and the small sciatic nerve are the collateral branches of the sacral plexus. The sciatic nerve is the terminal branch of the plexus and will be discussed separately in detail.

The *nerve to the quadratus femoris and gemellus inferior* arises from the anterior divisions of the fourth and fifth lumbar and the first sacral spinal nerves. It leaves the pelvis through the greater sacrosciatic foramen and descends beneath the sciatic nerve, gemelli muscles and the tendon of the obturator internus.

The *nerve to the obturator internus* arises from the anterior divisions of the fifth lumbar and first and second sacral spinal nerves. It leaves the pelvis through the greater sacrosciatic foramen below the pyriformis. It re-enters the pelvis through the lesser sacrosciatic foramen and ends within the obturator internus muscle. It gives off a branch to the gemellus superior muscle.

The *nerve to the pyriformis* arises from the posterior divisions of the first and second sacral spinal nerves. It innervates this muscle by entering its anterior surface.

The *superior gluteal* nerve arises from the posterior divisions of the fourth and fifth lumbar and first sacral spinal nerves. It leaves the pelvis through the greater sacrosciatic foramen above the pyriformis muscle in company with the gluteal vessels. The nerve divides into a superior and an inferior branch. The former accompanies the deep gluteal artery and ends in the gluteus minimus muscle. It also gives off a branch

to the gluteus medius. The inferior branch accompanies the lower branch of the gluteal artery across the gluteus minimus and supplies that muscle and the gluteus medius. It ends in the tensor fasciae femoris.

The *inferior gluteal* nerve arises from the posterior divisions of the fifth lumbar and first and second sacral spinal nerves. It leaves the pelvis through the greater sacrosciatic foramen below the pyriformis muscle. The nerve has a curved course with a superior concavity and divides into many branches which penetrate the gluteus maximus muscle.

The *small sciatic* nerve arises in part from both the anterior and posterior divisions of the first, second and third sacral spinal nerves. It leaves the pelvis through the greater sacrosciatic foramen below the pyriformis muscle. It descends beneath the gluteus maximus muscle with the sciatic artery and passes down the posterior surface of the thigh beneath the fascia lata. It continues to the lower part of the popliteal space, passing over the long head of the biceps femoris muscle. It pierces the fascia at that point and accompanies the external saphenous vein. The branches of this nerve are all cutaneous. The gluteal branches turn upward around the lower border of the gluteus maximus to supply the skin covering the lower and outer part of that muscle. The perineal branches are distributed to the skin of the upper and inner side of the thigh on its posterior surface. One of the branches, longer than the others, the *inferior pudendal*, curves forward beneath the superficial fascia of the perineum and is distributed to the skin of the scrotum in the male and the labium major in the female. The femoral branches descend and are distributed to the back and inner sides of the thigh to the skin over the popliteal space and to that over the upper part of the leg.

Physiology of the Sacral Plexus

The *gluteus maximus* muscle is a powerful extensor of the thigh upon the pelvis and of the pelvis upon the thigh. It also produces outward rotation of the thigh.

Contraction of the *gluteus medius* muscle in its entirety produces direct abduction of the thigh. Isolated contraction of the anterior fibers produces internal rotation with abduction and slight flexion while contraction of the posterior fibers causes external rotation with abduction and extension. It also aids in fixation of the pelvis.

The *gluteus minimus* has the same action as the gluteus medius.

The *tensor fasciae femoris* flexes and internally rotates the thigh. It also tenses the external part of the femoral fascia.

The *pyramidalis, gemelli, quadratus femoris and obturator internus* muscles are all external rotators of the thigh.

Surgery of the Sacral Plexus

The surgical lesions of the sacral plexus and its collateral branches are similar in extent and nature to those described under the lumbar plexus.

Chapter XXXIV

THE FEMORAL NERVE

Injuries to the femoral nerve were observed in the War in less than 1 to 2 per cent of the cases studied. They occurred as the result of stab and gunshot wounds, fractures about the



Fig. 294 Injury to femoral nerve with sensory loss over musculocutaneous.

pelvis, and aneurysms of the femoral artery. In civil life, tumors, tuberculosis, psoas abscess, reduction of congenitally dislocated hips, operative wounds, mononeuritis and prolonged gynecologic operations with the thighs strongly abducted, have caused its paralysis. When injured at the highest part of the thigh, below Poupart's ligament, all the terminal branches of the nerve are affected, and in addition to the quadriceps, other muscular branches are affected. Inasmuch as the terminal branches are immediately dispersed, commonly it is the nerve to the quadriceps or the internal saphenous which is injured. In none of our cases were all the branches involved. In the majority only the branch to the quadriceps was injured. When the long saphenous alone was injured no muscular paralysis occurred (Fig. 294).

Motor Symptoms

When the paralysis includes all of the branches, the hip joint can not be flexed and the leg can not be extended. When

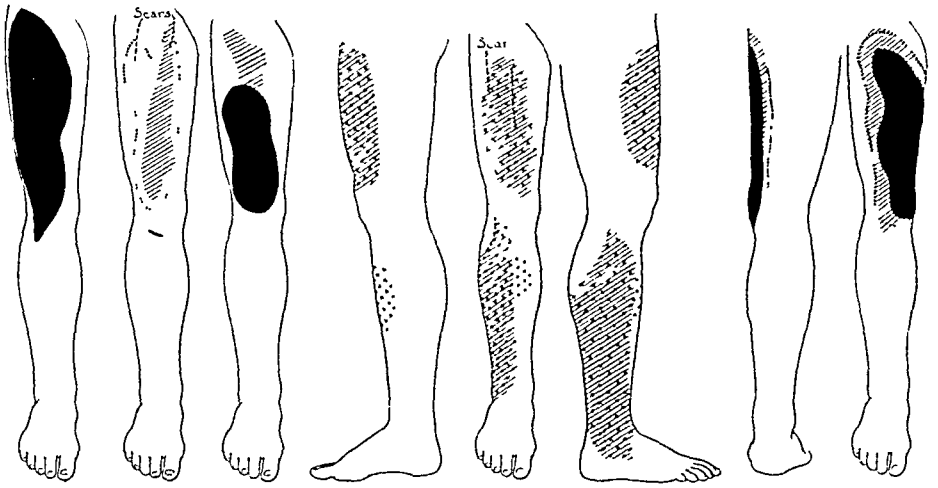


Fig. 295. Sensory loss in femoral nerve lesions.

only the terminal branches to the extensors of the leg are paralyzed, the patient walks in a manner to avoid bending the knee. Walking is extremely difficult and frequently the leg gives way at the knee, causing the patient to fall. Often the patient walks backward, attempting to produce a hyperextension at the knee to prevent the leg from giving way. The lower extremity is often flexed at the hip, then flung forward and slapped down. This gives the appearance of a pseudo-steppage gait. At the same time the trunk is bent forward and the uninjured extremity is advanced by a short step. Often assistance in keeping the leg extended is rendered by pressure backward upon the thigh by the hand of the affected side. The knee jerk is diminished or absent.

Atrophy is seen over the thigh and the markings of the thigh brought about by the tensing of the aponeurotic covering when the quadriceps contracts are missing.

Sensory Symptoms

The sensory changes vary. There may be a loss over the outer surface of the thigh and the inner surface of the leg.

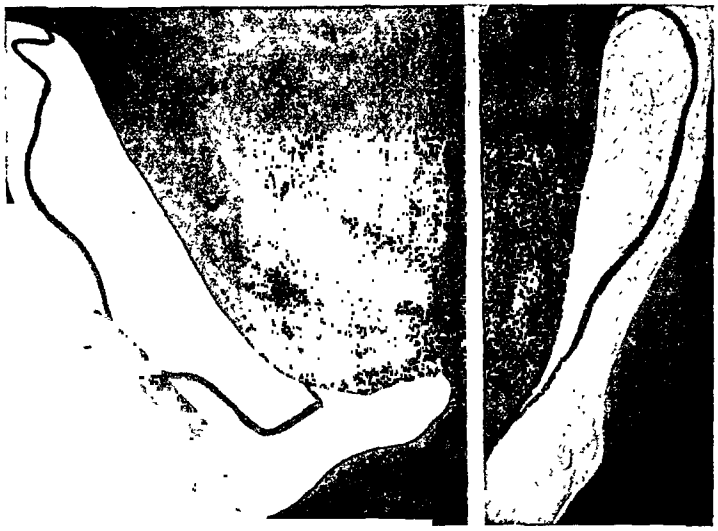


Fig. 296. Injury to long saphenous nerve.

However, in the majority of our cases only the motor branch to the quadriceps muscle was injured and the sensory loss corresponded to the area of the middle and internal cutaneous branches (Fig. 295).

Signs of Recovery

The general tendency in our cases was toward spontaneous recovery, and none of the cases came to operation. Disability in walking persisted after return of function in the muscles and probably was due to weakness or distention of the aponeurotic covering of the knee joint, as has been pointed out by Meige.

Long Saphenous Branch

The *long saphenous* nerve is more often involved than the other terminal branches and produces a loss of sensation on

the inner surface of the leg. Subjectively, sharp pain may occur along its distribution. Usually the pain subsides, and in only one case, in which the sciatic nerve was also injured, was there severe pain of a causalgic nature sufficient to warrant operative interference (Fig. 296).

The femoral nerve arises from the anterior divisions of the second, third and fourth lumbar nerves and is the largest branch of the lumbar plexus. Occasionally it may receive an additional contribution from the first or fifth lumbar nerves. It passes downward through the fibers of the psoas muscle, emerges from within the substance of the muscle at the lower part of its lateral edge and continues downward between it and the iliacus muscle. The iliac fascia separates the nerve from the peritoneum and the cecum on the right and the colon on the left. The nerve passes beneath Poupart's ligament where it is separated from the femoral artery by the psoas minor muscle. As it enters the thigh it becomes flattened and divides into an anterior and posterior division (Fig. 297).

Within the abdomen the femoral gives off three to four muscular branches to the iliacus muscle and a small branch to the inferior portion of the psoas muscle. A variable number of branches supply the femoral artery which are distributed to the upper portion of that vessel. Beneath Poupart's ligament the nerve lies above and lateral to the femoral artery.

From the anterior of the terminal divisions of the nerve arise the *middle* and *internal cutaneous* nerves and *muscular branches*. The middle cutaneous nerve pierces the fascia lata about 3 inches below Poupart's ligament and descends along the anterior surface of the thigh to supply the skin and subcutaneous tissue as low as the knee. Here its branches anastomose with those of the internal cutaneous and the long saphenous nerve to form the patellar plexus. The internal cutaneous passes obliquely across the sheath of the femoral artery and divides into an anterior and posterior branch. The former descends on the sartorius muscle and perforates the fascia lata at the lower third of the thigh. Here it divides into

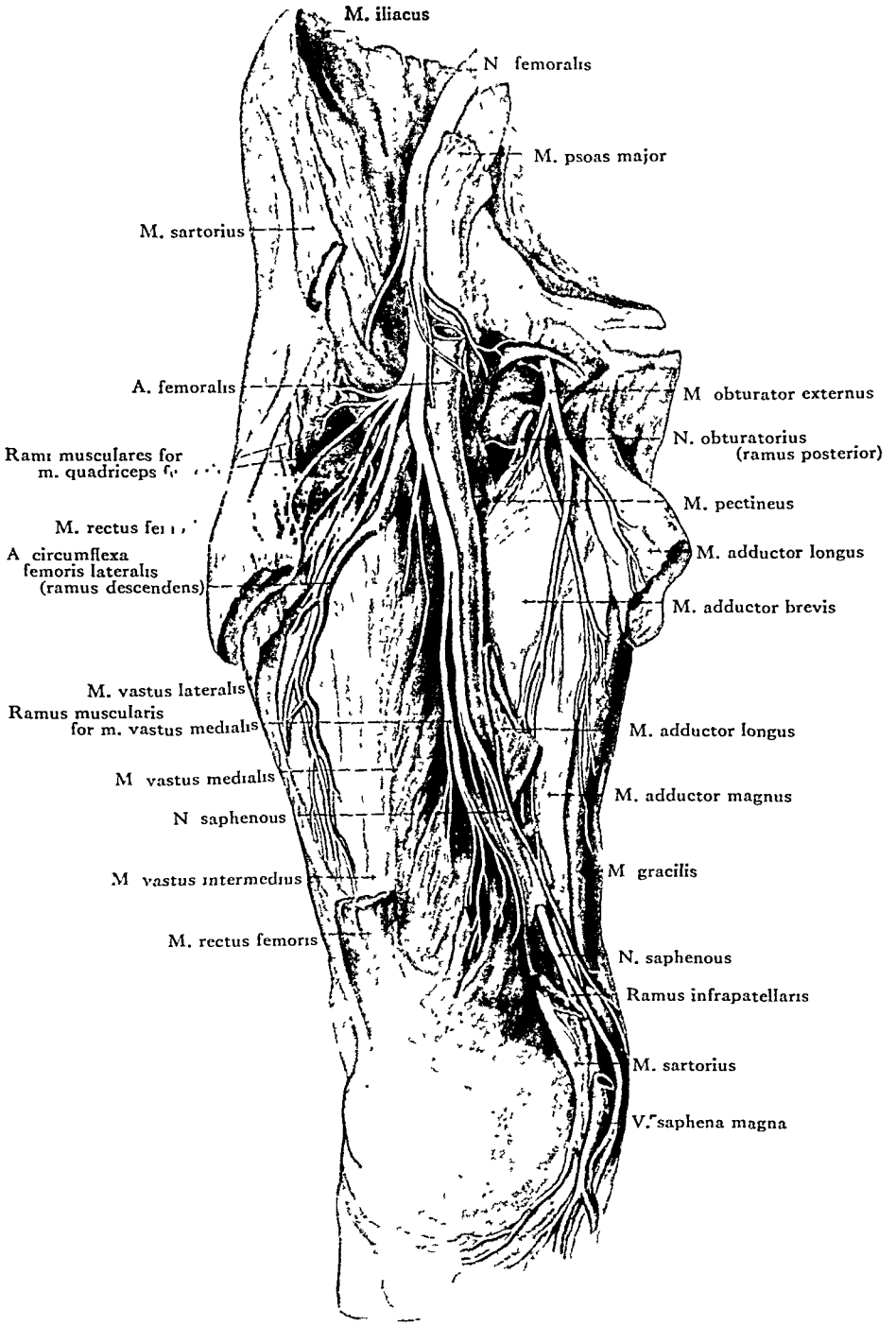


FIG. 297. Drawing to illustrate anatomy of femoral nerve.

a branch which supplies the skin on the inner side of the thigh as low as the knee. The posterior branch descends along the inner border of the sartorius muscle to the knee where it pierces the fascia lata and then continues downward to supply the skin of the inner aspect of the leg. The muscular branches supply the pectineus muscle just below the inguinal ligament. A second branch supplies the sartorius muscle at the level of the origin of the middle cutaneous nerve.

The *long saphenous* nerve is the largest cutaneous branch of the femoral and arises from its posterior terminal division. It lies in front of the femoral artery beneath the aponeurosis of Hunter's canal as far as the opening in the lower part of the adductor magnus muscle. It then passes medially, leaving the course of the artery, and descends along the inner aspect of the thigh beneath the sartorius muscle. It pierces the fascia lata between the sartorius and gracilis tendons and becomes subcutaneous. The nerve then continues distally along the inner side of the leg, accompanied by the internal saphenous vein. It passes behind the internal border of the tibia and at the lower third of the leg divides into two branches. One of these continues along the margin of the tibia to terminate at the ankle while the other passes in front of the ankle and is distributed to the skin along the inner side of the foot as far as the great toe. (Fig. 298.) A rather large patellar branch supplies the skin in front of the patella and helps form the patellar plexus. The muscular branches of the posterior division of the femoral nerve supply the four portions of the quadriceps extensor muscle. The branch to the rectus femoris is given off high in the thigh and enters its under surface. A small filament of this branch supplies the hip joint. The branch to the vastus externus is large and follows the course of the descending branch of the external circumflex artery to the lower portion of the muscle. It also supplies a twig to the knee joint. The branch to the vastus internus is long and descends with the long saphenous nerve along the external side of the femoral vessels. It enters the muscle at its middle and it too supplies a

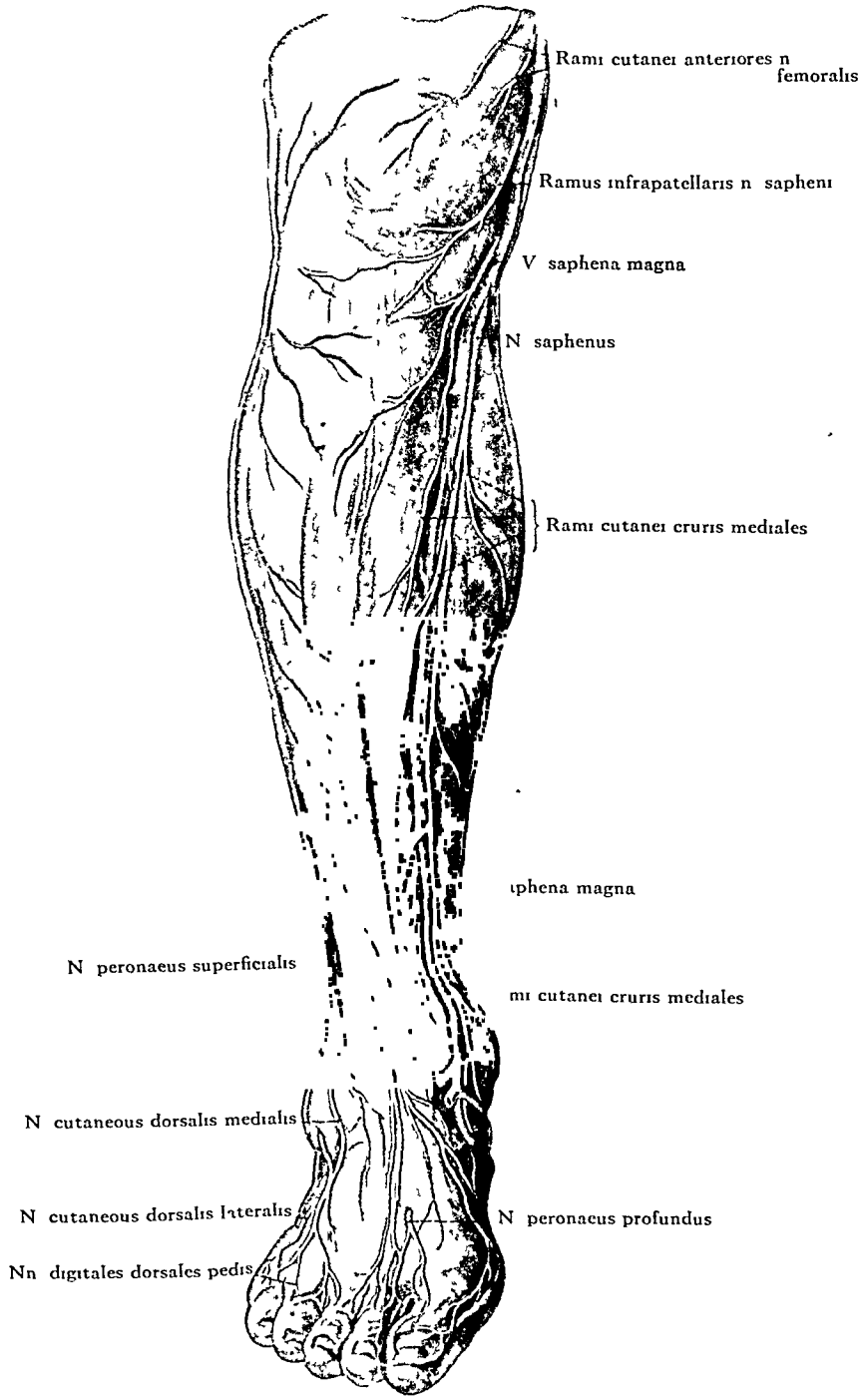


FIG. 298. Drawing to illustrate anatomy of cutaneous nerves of lower extremity.

filament to the knee joint. The nerves to the vastus intermedius are several in number and enter the muscle on its anterior surface at about the middle of the thigh. A twig from this nerve also supplies the knee joint.

Physiology

The femoral nerve supplies the muscles of the anterior femoral region. These are the *sartorius* and the components of the *quadriceps extensor* (rectus femoris, vastus externus, vastus internus, vastus intermedius and the subcrureus). The sartorius muscle primarily flexes the leg upon the thigh but as it continues its action it flexes the thigh upon the pelvis and rotates the thigh outward. When the knee is bent, the sartorius assists the semitendinosus and semimembranosus to rotate the tibia internally.

The quadriceps extensor group extends the leg upon the thigh. In addition the rectus femoris assists the iliopsoas to support the pelvis and trunk on the femur. It also assists in flexing the thigh on the pelvis or if the thigh is fixed it will flex the pelvis. The vastus internus draws the patella upward and inward.

Surgery

The femoral nerve may be injured in fractures of the pelvis particularly if the ascending ramus of the pubis is broken. Fracture of this bone usually occurs near the point where the nerve crosses the bone. It may be injured also in fractures or dislocations of the femur or it may be compressed by pelvic tumors which grow downward along the inguinal canal. Because of its superficial position and its close relation to the femoral vessels it is exposed to stab wounds of the groin. Of course, a wound of any severity in this region is very apt to cause death from hemorrhage or make an amputation necessary. As it enters the thigh the nerve lies externally and above the artery and vein so that the position of the structures from the lateral to the medial sides is nerve, artery and vein. In

certain instances an extensive laceration of the muscles with subsequent scar formation may compress the nerve severely.

It is almost impossible to suture the nerve in the thigh because it breaks up into so many smaller branches so soon after its appearance in the thigh. Other steps such as direct neurotization of the muscles by implantation of the central ends may prove valuable. However, it may be necessary to overcome the disabling deformity of complete paralysis of these muscles by tendon transplantation.

Chapter XXXV

THE CRANIAL NERVES

Few of the cranial nerves are injured alone. Intracranial injury may occur along with more serious injuries to more important structures and early death makes it impossible to



Fig. 299.



Fig. 300.

Fig. 299. Usual limit of sensory loss in complete division of sensory root of trigeminal nerve.

Fig. 300. Deviation of jaw toward paralyzed side in a lesion of motor root of trigeminal nerve.

note their occurrence in warfare. We have observed a small number of lesions of the branches of the fifth nerve. Rarely have we observed an isolated complete paralysis of the seventh nerve. There have been many more cases of isolated injury to the spinal accessory nerve. The glossopharyngeal and hypoglossal nerves were injured together not infrequently in extracranial injuries. These multiple cranial nerve lesions constitute an interesting chapter of the injuries to the cranial

nerves in the World War and will be described in full in the next chapter. We will, therefore, briefly describe the clinical symptoms of injury to the trigeminal, facial and spinal accessory nerves. Of these the facial nerve offers the only practical surgical problem for restoration of its function.

Trigeminal Nerve

Lesions of the trigeminal nerve, while not uncommon, usually occur in one of its sensory branches. (Fig. 299.) Occasionally the motor branch has been included in the injury. At times, the masseter alone or together with the pterygoid and temporal muscles was found paralyzed. In the cases observed by us no loss of taste in the anterior two-thirds of the tongue could be demonstrated. Often the other wounds about the head were so serious that little or no attention was paid to the possible injury of the branches of the fifth nerve. When observed many months after injury in base hospitals, the number of cases which showed a residue of serious injury to this nerve was hardly occasional. When the masseter was paralyzed no tensing of the muscle occurred upon tightly clenching the jaws; and chewing was difficult on that side. In paralysis of the pterygoid opening the jaws and protruding the lower jaw was accompanied by deviation of the lower jaw to the paralyzed side (Fig. 300). There was no indication for surgical interference in any of our cases.

Facial Nerve

Far more frequent were the injuries to the seventh nerve. Here, too, injury to some of the branches was more frequent than injury to the trunk of the nerve. As a rule, such injuries were only incidental to the general mutilation of the face. Rarely was the nerve found to be injured before its exit from the stylomastoid foramen. It is conceivable that it was so injured in many fatal cases of basilar skull fracture and other intracranial injury. It was often found to be damaged in wounds of the parotid region and of the neck. It is notable,

however, that in the material seen numbers of months after injury very few cases of facial palsy were seen.

Wounds of the cranium, particularly in the temporofrontal



FIG. 301. Paralysis of facial nerve.

region, are often accompanied by paralysis of the motor filaments of the orbicularis palpebrarum, or the frontalis muscles. Most frequently some of the branches of the seventh nerve are injured alone. Paralysis of the trunk of the seventh nerve is characterized by complete absence of movements of expression on the paralyzed side, with inability to wrinkle the forehead upward or to close the eye. Attempts to close the eye are associated with a movement of the eyeball upward. This is Bell's phenomenon. In addition to this, the patient is unable to pucker his lips, to smile or show the teeth on the paralyzed side. The expression of the face is characteristic. The paralyzed side is "washed out" and expressionless, and because of atony the angle of the mouth droops, the nasolabial fold is obliterated and there is an effacement of wrinkles on the forehead. Frequently, because of inability to hold the lips closely together, there is drooling of saliva. All of the signs are exaggerated during attempted movements of the face, or during emotional expression, as in laughing (Figs. 301 and 302). When injured within the bony canal before its exit from the stylomastoid foramen, taste is lost over the anterior two-thirds of the tongue.

Frequently the trunk may be only partially injured, and in addition to the preservation of reaction of the muscles to faradic stimulation more than fourteen days after injury,



A B C

FIG. 302. Paralysis of facial nerve (A) at rest; (B) showing teeth; (C) closing eyes.

certain motor signs indicate the recoverability of the lesion. Not only can the eyelids of the affected side be closed more completely than in the case of total paralysis, but twitches can be observed in the lower lid accompanying the winking of the opposite eyelids consequent to threatening gestures. The nasolabial fold is not completely obliterated. The lips on the affected side are not separated and the lower lip upon attempts to show the teeth, although not moved away from the midline, show vertical wrinkling. Even in the presence of a complete reaction of degeneration, these signs may justify us in predicting spontaneous recovery of the nerve.

As in civil life so in military practice it is notable that following incomplete recovery contracture and overaction of parts of the muscles supplied by this nerve occur. Upon closing the lids or winking contraction of the muscles of the lower parts of the face may be observed. Even insignificant injuries to branches to this nerve below the eye are followed by the persistence of muscular twitching coincident with winking. Conversely, in showing the teeth the eyelids of the affected side are more closely approximated.

Anatomy of the Facial Nerve

The facial nerve has its cells of origin in the reticular formation of the lower part of the pons. The fibers pursue a curved course within the pons. They first pass backward and inward and then turn upward and forward to form an eminence on the floor of the fourth ventricle. Finally they bend downward and outward at a sharp angle around the nucleus of the sixth cranial nerve and leave the brain stem at the cephalad border of the medulla.

The acoustic nerve lies lateral to the facial and between them is the nervus intermedius of Wrisberg. This latter may be regarded as the sensory root of the facial nerve. Its cells of origin lie within the geniculate ganglion.

The facial nerve is firmer, rounder and smaller than the acoustic nerve. It passes forward and outward to enter the internal acoustic meatus with the acoustic nerve. At this point the facial lies over the acoustic in a groove formed by the latter nerve. The nervus intermedius lies between them. They have a common sheath which is formed by continuations of the meninges and of the subdural and subarachnoid spaces all of which reach to the bottom of the meatus. At that point the facial enters the facial canal (aquaeductus fallopii) and follows its course through the petrous portion of the temporal bone to the stylomastoid foramen. At first, directed outward between the cochlea and the vestibule toward the inner wall of the tympanum, the canal bends backward and arches downward behind the tympanum to the stylomastoid foramen. At the point where the nerve changes its direction there is a greyish red swelling, the geniculate ganglion.

After its exit from the stylomastoid foramen the facial runs forward in the substance of the parotid gland, crosses the external carotid artery and divides behind the ramus of the mandible into its two primary branches, *temporofacial* and *cervicofacial*. As these two branches and their ramifications separate from each other they present the appearance of a bird's claw (Fig. 303).

In its course the facial forms many communications. Within the internal acoustic meatus there is a small twig which joins the acoustic nerve. Several communicating branches arise from the geniculate ganglion. The great superficial petrosal nerve joins the sphenopalatine ganglion to form its motor nerve. The small superficial petrosal nerve communicates with the otic ganglion. The external petrosal joins sympathetic fibers which accompany the middle meningeal artery. Within the facial canal a communicating twig joins the nerve from the auricular branch of the vagus. After its exit from the sytlomastoid foramen the facial sends a small branch to the glossopharyngeal and another to the vagus. At the same level communicating branches join the great auricular nerve of the cervical plexus; the small occipital nerve behind the ear; and the terminal branches of the three divisions of the trigeminal nerve. In the neck the facial communicates with the transverse cervical nerve.

Several larger branches of distribution arise from the facial in its course. The tympanic branch arises opposite the pyramid, passes through a small canal in the bone and supplies the stapedius muscle.

The *chorda tympani* is given off about a quarter of an inch above the stylomastoid foramen. This nerve passes from below upward and forward within a distinct canal. It enters the tympanic cavity through an opening on its posterior wall between the opening of the mastoid cells and the attachment of the tympanic membrane. Here it is covered by mucous membrane. The nerve passes through the tympanic cavity and over the handle of the malleus. It leaves the cavity through a foramen at the medial end of the fissura petrotympanica (Glaseri). It then descends between the two pterygoid muscles, meets the lingual nerve at an acute angle and accompanies it to the submaxillary gland. Part of the nerve joins the submaxillary ganglion while the major portion continues through the muscular substance of the tongue to the mucous membrane which covers its anterior two-thirds.

(*To Be Concluded*)

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DISCUSSION OF THE DIAGNOSIS AND X-RAY TREATMENT OF MALIGNANT DISEASE OF BONE*

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MUCH of the substance of this paper is at variance with accepted views and will be disagreed with by many readers as it departs from orthodoxy at several points. The subject of bone tumors is far too obscure and unnecessarily so. It stands greatly in need of simplification.

The skeleton is a peculiarly suitable structure for investigation by *x*-ray methods. Since their advent into medicine, knowledge of the skeleton, its anatomy, physiology and pathology has grown enormously. The skeleton is far better understood than connective tissue, cartilage, adipose tissue, muscle, etc.

Of the diseases of bone, which have been most extensively studied, both on account of the great aid afforded by *x*-ray methods and because of their highly lethal nature, tumor has attracted much attention. Bone tumor has been for the past several years a popular subject for investigation and publication. Impetus to this arises from the work of the Registry of Bone Sarcoma of the American College of Surgeons. Credit for the notable accomplishment of this body should be given to Codman.

There are such authoritative publications on malignant bone tumors that any prospect of adding anything original to that which has already been written is almost out of the question. However, many physicians are unacquainted with this work. It will be the endeavor of this paper to direct attention to and popularize some of the excellent existing publications on malignant bone tumors with the idea of simplifying this obscure subject.

These publications are the studies of the material in the Registry of Bone Sarcoma of the American College of Surgeons by Codman[1] and Kolodny,[2] various publications by Bloodgood,[3] Ewing,[4] and Connor,[5] and the monograph of Geschickter and Copeland.[6] All of these are masterly. Nevertheless, it seems to a radiologist that there are certain points concerning bone tumors that these writers fail to develop. I wish to call attention to a significant fact, i.e., that these publications, with the exception of those of Ewing, were written by surgeons, and they necessarily lack the general view of the subject of gross bone pathology which one acquires in the radiological or pathological laboratory. This applies equally to matters of incidence, differential diagnosis and treatment.

INCIDENCE

The incidence of malignant bone tumors is not commensurate with the amount that has been written on the subject. The writings incline one to believe that malignant disease of bone, unfortunately too common as it is, is commoner than is actually the case. Furthermore, the more

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important publications have been made by men who have had an immense amount of accumulated material with which to work.

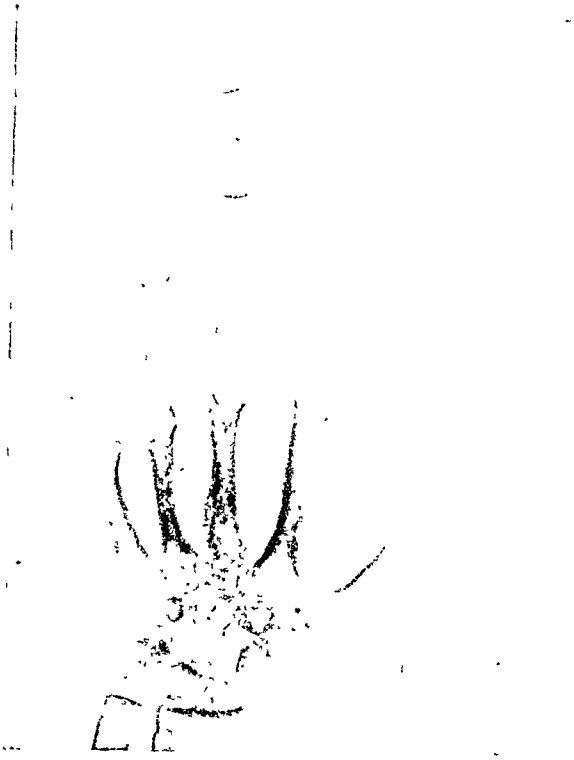


FIG. 1. Radiograph of hand made Nov. 5, 1928. Characteristic endothelial myeloma of third metacarpal bone. Excision of metacarpal.

One result of this condition has been the elaboration of classifications, etiological theories, and the promulgation of certain ideas in regard to treatment of bone tumors which are of theoretical rather than practical use. Unfortunately, the task of the physician or radiologist dealing with the occasional case of malignant disease of bone has not been made proportionately easier.

After all is said and done, the greater proportion of individuals with malignant condition in the skeleton will have to be cared for by what may be termed the casual radiologist or surgeon. All such cases cannot be put under the care of what may be termed specialists in the field of malignant bone disease. Except in those centers where special attention is paid to malignant bone tumors, and hence where many patients apply for treatment, their occurrence is fortunately rather rare. In

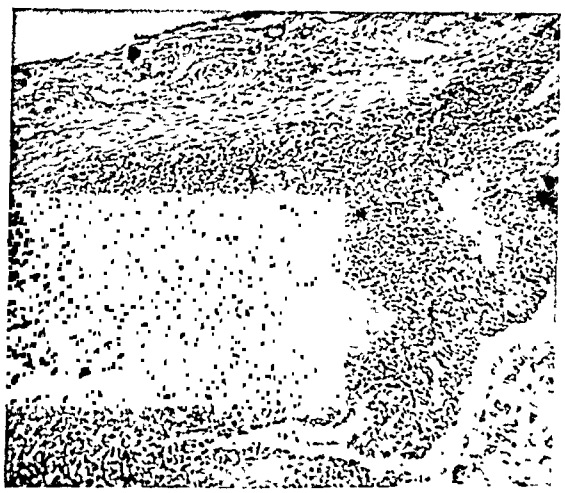


FIG. 2. Section of tissue near metacarpal taken at operation. Shows solid invasion of soft tissue by tumor cells. There is no intercellular substance and there is a tendency toward perithelial arrangement. There are several large blood spaces lined with tumor cells.

the hospitals allied to the Washington University School of Medicine, the Barnes Hospital, the St. Louis Children's Hospital, the St. Louis Maternity Hospital, in a period of eighteen years and in 123,285 hospital admissions there have been 220 cases of sarcoma with 96 examples of primary malignant bone tumors. Over the same period there were in the Barnes Hospital, 277 cases of secondary or metastatic malignant involvement of the skeleton in 5883 cases of carcinoma. The other tumors capable of skeletal metastasis have been too few in number and uncertain in diagnosis to warrant the labor of segregating them and forming an additional classification. The commonest sites of origin of these carcinomata were:

Alimentary tract	1214
Uterus	865
Breast	352
Prostate	234

The remaining cases were widely scattered among other epithelial structures.

The ratio of incidence of metastatic to primary bone tumor in the preceding statistics is 2.9 of the former to 1 of the latter. High as the figure is for metastatic bone tumor, I doubt if it shows the true incidence of that complication as such skeletal foci are not sought for as intensively as are the primary bone tumors. Systematic skeletal radiography in cancer would undoubtedly greatly increase the foregoing ratio. Our views of the secondary and remote manifestations of cancer are predicated on the findings in the autopsy room. These views should be modernized and brought into correct relationship with the facts as developed by radiography. It is not sufficiently well known how searching is radiography nor the extent to which it is capable of supplementing the knowledge attainable from the postmortem examination. In the vast majority of examples of metastatic bone tumors this unfortunate happening is of later occurrence in the course of cancer. With the increasing efficiency of treatment of cancer late manifestations are becoming more of a problem than ever before.

DEFINITIONS OF TERMS AND CLASSIFICATIONS

In view of this, some definitions of terms and a classification are in order, to secure an intelligent approach to the problem of differentiation of primary and metastatic skeletal involvement in malignant disease. This is an essential preliminary to the correct treatment of the cases with skeletal neoplastic disease. The classification and terms applied to *all bone lesions* by the Registry Committee of the American College of Surgeons is as follows:*

1. Metastatic tumors primary in tissues other than bone
2. Periosteral fibrosarcoma
3. Osteogenic tumors: (a) benign, and (b) malignant
4. Inflammatory conditions
5. Benign giant cell tumors
6. Angiomata: (a) benign, and (b) malignant
7. Ewing's tumor

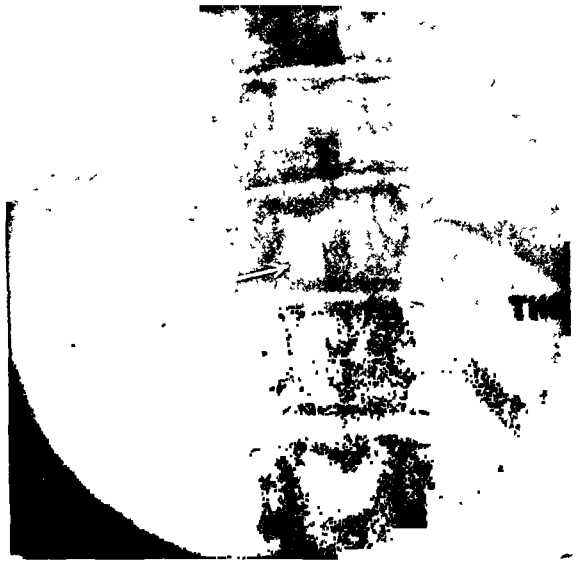


Fig. 3. Radiograph of spine of Feb. 26, 1932. Arrow indicates destructive lesion in vertebra. Since first observation forearm amputated for local return of tumor.

8. Myeloma. (Among other omissions fracture and metabolic disease of bone!)

It should be said that in the radiological laboratory infectious processes, or their result in bone, offer great difficulty in differential diagnosis. Such differentiation is too lengthy to be included in the limits of this paper. Kolodny modifies the foregoing classification as far as it applies to primary malignant bone tumors as follows:

1. Osteogenic sarcoma
2. Ewing's sarcoma
3. Myeloma
4. A group of unclassified sarcoma, including among others angioendothelioma and extraperiosteal sarcoma.

This classification is admirable and it is especially applicable in the *x*-ray laboratory. It is also useful in the treatment of the diseases under discussion.

The writer differs with such an authority as Kolodny as to the classification of endothelioma of bone or Ewing's sarcoma, believing that it should be grouped with

* Taken from Kolodny, A., Bone Sarcoma. Chicago, Surgical Pub. Co., 1927.

the myelomas. Two of the cases observed by the author (Figs. 1–7), indicate by their course and spread that the tendency of throughout the marrow system, and the tumors not attaining great size, does not come to the notice of the surgeon and

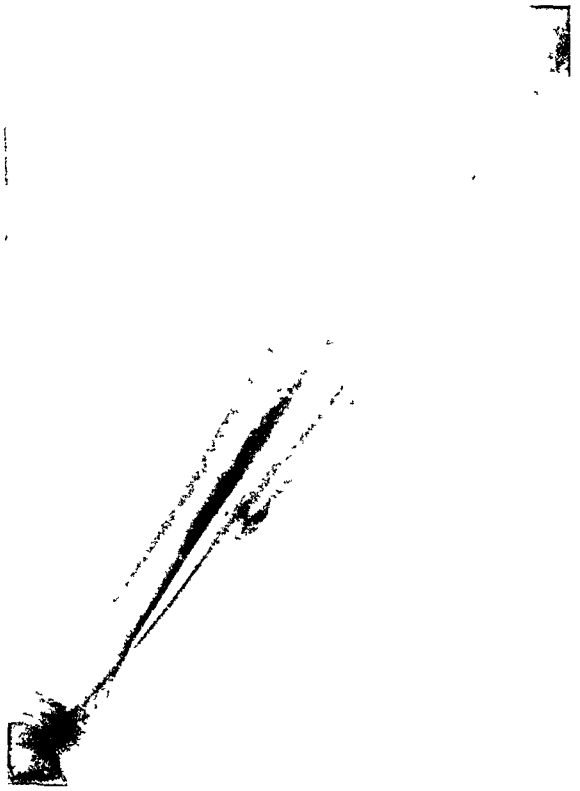


FIG. 4. Lateral radiograph of right leg of second case, April 3, 1929.



FIG. 5. Lateral radiograph of right leg, Aug. 23, 1929, showing characteristic effect of irradiation of Ewing's tumor.

endothelial myeloma is to the progressive involvement of the entire marrow with the formation of large osteolytic central tumors in multiple regions of the skeleton. In the writer's view they progressively involve the marrow system equally with the other myelomas. It would seem that the chief difference between endothelial myeloma and those of other type is that in the former the primary tumor attains a larger size at an earlier period than is the case in the latter, and, hence, attention is called to it sooner. In the published cases of endothelial myeloma there is remarkable paucity of reports as to the condition of the remainder of the skeleton at the time of discovery of the tumor. There is also a great lack of knowledge of the progression or course of the disease in a large group of cases. Myeloma of other type developing rapidly radiologist until it is widespread. Apparently the reverse is true in endothelial myeloma; it is discovered earlier in its life cycle than the other myelomas.

In spite of the great merit of Kolodny's classification of the malignant bone tumors, it has seemed to the writer that this difficult subject could be further simplified by the following classification:

1. Tumors which originate from the tissues housed in bone. These are the various types of myeloma including endothelial myeloma (Fig. 8). Epithelial tumors of dental origin in the maxillae belong in this heading but are only mentioned. There must necessarily be a few rare tumors of blood vessel origin and malignant nature, angioendothelioma, etc., which belong in this class.

2. Bone tumor proper. Osteogenic sarcoma (Fig. 9).

3. Tumors originating from the invest-



Fig. 6. Anteroposterior radiograph of left knee joint of Oct. 8, 1929. Arrows indicate bone defects in femur and tibia At this period there was a huge defect in the skull.

ments of bone, the periosteal fibrosarcoma, parosteal sarcoma, capsular sarcoma (Fig. 10). These last named are in no sense true bone tumors, except from the accident of juxtaposition. Only rarely do they involve, extend to, or infiltrate bone. They may, however, on occasion, cause resorption of bone which seems to be a pressure effect arising from their increase in size.

METASTASIS

Some remarks on the metastasis and spread of primary bone tumors are perti-



Fig 7. Section from rib removed at autopsy. Pathological picture closely resembles that of Figure 2.



Fig 8 Lateral radiograph of foot with characteristic changes in plasma cell myeloma in distal portion of a limb

nent at this point.

In spreading, sarcomas follow the path of least resistance, and those of the skeleton are no exception to this rule. Osteogenic

sarcoma extends far beyond the limits which are demonstrable by *x*-ray or other methods of examination of the affected



Fig. 9.

bone. It has been shown that it may extend far up or down the marrow cavity. Large blood spaces develop in them, and these become invaded by tumor cells. Osteogenic sarcomas also follow to the limits of their extent the loose areolar tissue spaces in intermuscular septa, around blood vessels, etc. In the extremities it doubtless early reaches the point where the septa are fused into the dense fascias at the termination of the segments of limbs. When metastasis takes place, it is by a blood stream distribution of the metastatic material. Therefore, pulmonary metastases are the first to occur. This occurrence is of such constancy that it can be stated as a general rule in sarcoma. Sarcoma does not travel through the lymphatics, hence regional lymph nodes are usually spared.

Osteogenic sarcoma is rapidly invasive of surrounding soft tissues. Endothelial myeloma and myeloma are only slightly invasive when they break through the bone, and spread to adjacent tissues, the former more so than the latter. Pulmonary metastases are not found in the author's cases. Usually in the case of all types of myeloma, little if any of the tumor is found beyond the confines or immediate vicinity of the bone. Endothelial myeloma



Fig. 10. Radiograph of leg of Dec. 24, 1930. Large soft tissue tumor. Bone is unaffected.

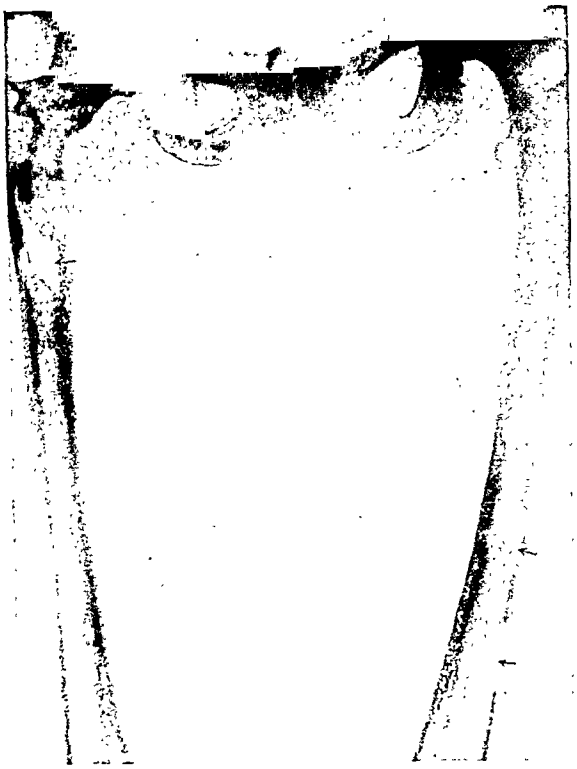


FIG. 11. Radiograph of femora. Arrows indicate destruction of bone by metastatic nodules of hypernephroma.



FIG. 12. Radiograph of chest with characteristic findings of metastatic pulmonary sarcoma.



FIG. 13. Lateral radiograph of leg of Nov. 11, 1931. Case of Paget's disease. Arrow indicates destructive process at upper end of tibia.



FIG. 14. Radiograph of chest of March 4, 1932. Arrows indicate areas of infiltration in lung thought to be a metastatic sarcoma from tibia.

while it may long remain a single focus is often said to metastasize to other bone. In the writer's opinion this merely represents a remote development of the same process in the marrow at a point distant from the first recognized growth. It is not a metastasis in the sense of embolic transport of malignant material from one point through the blood or lymph circulation to more remote ones elsewhere in the marrow.



Fig. 15. Photograph of gross material removed from tibia. White zone indicated by arrow was adenocarcinoma mocroscopically confirmed.

Extraperiosteal sarcoma, periosteal fibrosarcoma and capsular sarcoma are less distinctive in their radiological manifestations than osteogenic sarcoma. Their spread and metastasis apparently are the same as that of the soft tissue sarcomas which arise wholly independent of bone. I would condense the facts of the spread and metastasis of these tumors in the following way: Osteogenic sarcoma spreads rapidly into blood spaces and vessels (which it may form) along the areolar tissues, and for distances which cannot be suspected. It metastasizes by blood stream distribution and does not involve the lymphatics. The metastases are pulmonary. Endothelial myelomas and the other myelomas spread more slowly. Of these two groups only endothelial myeloma invades surrounding tissue to any great extent. All types of myeloma tend to involve ultimately the entire marrow system, with varying degrees of destruction of the containing bone. Extreme destruction of the marrow seems to be the cause of death. Myelomas do not metastasize as a general rule, certainly this is not observed in the *x*-ray laboratory.

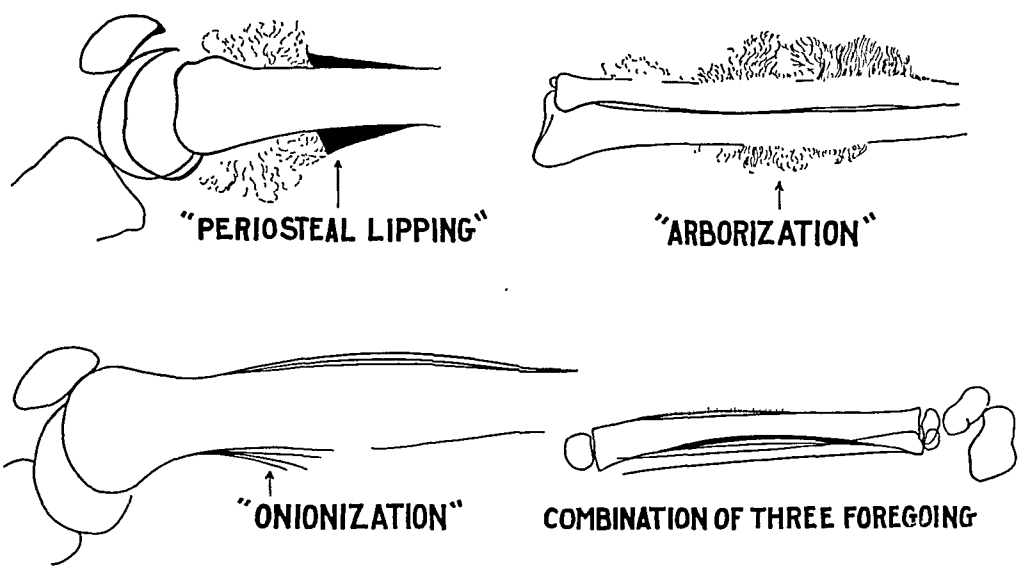


Fig. 16.

Sarcomas, either osteogenic or of other origin, only exceptionally extend to, that is, infiltrate adjacent bones or metastasize



Fig. 17. Example of tumor and pathological fracture in which amputation is indicated.

to remote portions of the skeleton. When bone metastases appear to be present the probabilities are that the tumor is of some other nature than sarcoma.

Giant cell sarcoma must be referred to here because it has been so long put in the same category as the sarcomas. This disease can be considered as a malignant one only when it is neglected. Correct treatment removes it from the class of malignant diseases. Giant cell tumor is only slightly invasive of surrounding parts, does not metastasize and it only rarely recurs after excision or amputation if properly done, and does not directly destroy life. It is to be borne in mind, however, that malignant bone tumors sometimes contain giant cells, and lead to mistakes in diagnosis. The giant cell tumors will be referred to again when the therapy of malignant bone tumors is considered.

Metastatic hypernephromas cause much confusion in the diagnosis of bone lesions, and, hence, in treatment. They may form



Fig. 18. Huge giant cell tumor too large for treatment other than amputation.

a single focus in a single bone, or they may have a wide distribution throughout the skeleton (Fig. 11) and at the same time the lungs may be the site of metastases. As far as radiological methods are concerned, they may simulate osteogenic sarcoma precisely both in their supposed primary focus in bone and in pulmonary distribution. They can also arise in younger persons which is not usually the case with the majority of the metastatic skeletal tumors. However, if there are multiple skeletal foci, and characteristic findings in the lungs, one may be safe in concluding that the case is not a bone sarcoma, but a hypernephroma.

Epithelial tumors, metastatic to bone, have certain characteristics. Recognition of these aids materially in their separation

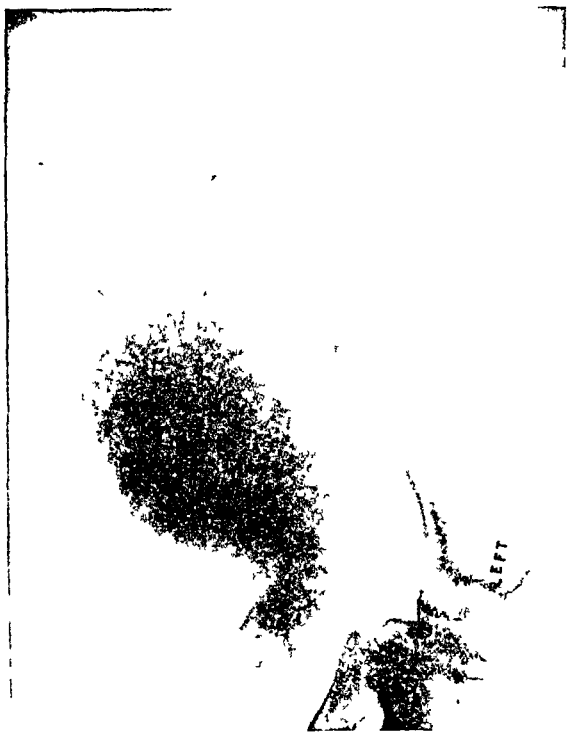


Fig. 19. Large parosteal sarcoma in which amputation is indicated. There is some evidence of pressure effect on posterior aspect of bone.

from primary malignant bone tumors. Obviously, the first point in separating primary bone tumors from epithelial metastases is that, in general, epithelial tumors will be found at a later age than is the case with sarcomas. If an epithelial tumor is metastatic to bone it is more likely to be multiple as to the bones involved. This is so contrary to the rule for bone tumor that it is a safe statement that multiple foci in bone eliminate primary bone tumor from consideration. Epithelial tumors so rarely metastasize distal to the most outlying lymph-node distribution, namely those at the elbow and knee joints, that it can be said that metastasis beyond these two joints is virtually never observed. Hence, a tumor arising in these distal segments of the limbs will not be a metastatic epithelial tumor. This particular behavior is also true of hypernephroma. Therefore, a tumor found in these locations must be primary in the bones involved and is either an osteogenic tumor or a myeloma. I will



Fig. 20.

digress enough at this point to say that in the examination of cases with actual or suspected bone tumor or metastasis that the entire skeleton be radiographed. This is a rule unfortunately more often observed in the breach than in performance. A radiological study in suspected cases of this nature is incomplete unless the chest is also examined.

Our material indicates that it is almost axiomatic that carcinomata do not metastasize to the lungs with the same manifestations as are found in sarcomas. That metastasis of the epithelial tumors to the lungs frequently occurs anyone with the

least knowledge of autopsy findings in carcinoma is well aware. This is most often by way of lymphatic permeation which



Fig. 21. Radiograph of knee joint, April 7, 1927. A giant cell tumor of inner condyle of femur.

does not produce the large globular growths (Fig. 12) in the parenchyma of the lungs usually found in metastatic sarcoma, or hypernephroma.

Bone sarcoma can occur late in life, but Kolodny (*loc. cit.*) is authority for the statement that 60 per cent of the cases reported have arisen on the basis of an existing Paget's disease. An example of such a case has been encountered which demonstrates that even so important a rule as this and the one of non-spread of epithelial tumors beyond knee and elbow joints, does admit of exception. (Figs. 13, 14 and 15.)

The spread and secondary manifestation of the other type of tumors in the skeleton and in the lungs have been too infrequent in the material studied to permit of any extended statement. Melanomas have never been noted metastatic in bone.



Fig. 22 Radiograph of same knee joint as in Figure 21, Dec. 7, 1929. Marked restitution and good function. Result of intensive x-ray treatment. Patient in satisfactory condition.

The majority of those arising in the skin seem to behave much as epithelial tumors do, and extend via the lymphatic system. Those from the nail bed and from the choroid seem to favor a blood-stream distribution to the parenchymatous organs. Tumors of the ovary and testes have been too infrequently noted in the *x*-ray laboratory and in character are too protean in their manifestation to permit of any general statement to be made concerning them

except that they destroy life before they involve the skeleton.

RADIOLOGY

It seems that those who write on bone tumors do so as if the subject were in a finished and complete state. The natural history of the disease, so to speak, appears to escape them. Because of this, overemphasis is put on certain things beyond their innate importance, while others are underestimated.

A radiologist can draw one conclusion in regard to much that has been written about bone tumors by those who are not radiologists. It is that instead of radiological findings contributing something essential to the final conclusion as to the condition in hand, that the history, gross pathology, and microscopic pathology are used to explain the *x*-ray findings. This is unfortunate, as unfortunate in fact as the case of the pathologist, who, given pathological material, must know the history, clinical course, etc., before he can venture upon an opinion as to the pathological material. Neither the pathologist nor the radiologist makes the final diagnosis. Their work is remarkably alike. Their service is to clarify and make certain, points in diagnosis and their function is to observe and interpret objectively material submitted to them for opinion. When the radiologist must have the complete clinical history before him, I am constrained to feel that his usefulness to those who have called upon him is at an end.

Writers on bone tumors are much given to stating that certain changes are pathognomonic of this, that or the other type of bone tumor. A small triangle of dense bone at the expanding portion of an osteogenic sarcoma has great importance paid to it as a diagnostic sign. "Arborization," i.e., the formation of fine lines of calcific material extending into the soft tissues, their course being perpendicular to the shaft of the bone, is also supposed to be pathognomonic of osteogenic sarcoma. The laminated structure in endothelial myeloma or "onionization" is also thought to be highly diagnostic (Fig. 16). The fact of the matter is, any of these three signs can accompany and do accompany conditions other than bone tumor. Single pathognomonic *x*-ray signs in bone tumors, either primary or secondary, are, in the writer's experience, non-existent. He feels that in the majority of instances an experienced radiologist can state the nature of a bone lesion with a correctness equal to that found in other medical opinion. However, he realizes his own incapacity to describe radiographic findings and likewise doubts the ability of others to do so. Identification of these conditions rests on the recognition of a congeries of physical changes in the involved bone, which elude description in words.

DIFFERENTIAL DIAGNOSIS

Unless the several malignant affections of the skeleton can be diagnostically differentiated, no intelligent method of dealing with them can be evolved. When confronted with the problem of a bone tumor it must be ascertained first whether it is primary or secondary. When this is achieved, what method of treatment is to be employed?

1. If the patient is a young person with involvement of the bones distal to the knee or elbow, and the tumor is single, we can assume that it is a primary tumor.

2. In the last condition, i.e., a single bone focus, proximal to knee or elbow joints, in a young person, and metastasis to the lungs, the condition may be either a primary bone tumor or a hypernephroma.

3. If there is a diffuse bone lesion involving many bones and indicating a centrifugal spread, one is not dealing with a primary bone tumor.

4. If one has a solitary bone lesion, even in view of a history of carcinoma or other malignant tumor, and there are round circumscribed areas of density in the parenchyma of the lungs, one is dealing with an osteogenic sarcoma or hypernephroma with metastasis in the lungs.

TREATMENT

In any bone lesion which is multiple, treatment resolves itself into the application of palliative measures. It is as a palliative measure that radiation therapy is of the most use. However, as it tends to lose its efficiency the longer it is applied, it should not be resorted to until symptoms are present. In other words, it should not be used as a placebo. Any skeletal lesion of the extremities which is definitely osteogenic sarcoma and not yet metastatic should in the opinion of this writer be treated with radical surgery, that is to say, that if the tumor is distal to the knee or elbow, amputation should be done, but, above the proximal joints. Osteogenic sarcoma of the humerus or shoulder girdle should have a shoulder girdle amputation. Since the spread of sarcoma may be of the nature pointed out earlier in this paper amputation at the hip joint for osteogenic sarcoma does not seem feasible, unless it be of the sclerosing type. The same statement is true of osteogenic tumors of the trunk, and in this field the writer believes that radiation therapy is the treatment of choice. Routine preoperative or postoperative radiation as a prophylactic measure in sarcoma has never had any special appeal to the writer and has only been given when requested by the surgeon.

In addition to the favorable response of endothelial myeloma to radiation and to the fact that there is ground for believing that it is a type of systemic disease it should be treated by radiation alone.

Where there are widespread metastases in osteogenic sarcoma, great pain, cachexia, etc., radiation should be applied. Pathological fractures of the extremities or great tumefaction, fungus formation, and ulceration should have in addition, and as a palliative measure, amputation or excision as the case may be, to be followed by radiation. (Figs. 17, 18, 19.) Surgery of this type has immense possibilities of affording symptomatic relief. It is not only justifiable but in the writer's opinion it is mandatory in those who suffer greatly. Not enough surgery of this type is done.

Secondary involvement of the skeleton with malignant disease presupposes, of course, widespread dissemination which makes surgical intervention quite out of the question on the face of it, unless it is palliative. These cases should be, and are, fit subjects for irradiation. It is the agency which gives them more relief, prolongs their lives, and contributes some degree of comfort. In the case of the spine there often is uncontrollable pain, even with morphine. Examples of relief from such pain by irradiation are within the knowledge of anyone with any material experience with the treatment of this type of case. Irradiation then is the symptomatic palliative treatment for widespread secondary tumors of the skeleton.

There have been enough examples of cure of giant cell tumor by irradiation that this agency should be the first choice in the treatment of these cases with certain exceptions. If irradiation after a reasonable trial fails to produce the desired result then one may resort to surgery. The exceptions are those where the tumor is of such great size and location that repair cannot be hoped for (Fig. 20). Irradiation should not be employed if a joint has been destroyed by the giant cell tumor and if recovery would result in a stiff flail or useless limb. In these last named conditions amputation is to be preferred. Admirable results are secured in giant cell tumor by irradiation alone (Figs. 21 and 22). It should be recalled that this tumor is not a common one, if it were there would be more cures reported.

The results reported by Coley following the use of his toxin treatment convince the writer that the toxin treatment of malignant bone disease should always be combined with the other methods of treatment.

What is to be expected from the treatment of tumors in bone? Of the myelomas with which the writer has had to deal a small amount of relief of pain has been

obtained almost immediately followed by development of secondary tumors elsewhere in the skeleton. The only example of osteogenic sarcoma that is known by the writer to have survived any method of treatment is one that he irradiated. This individual is alive nearly ten years after the development of the tumor. She was treated wrongly in every way. Amputation was recommended and refused. Biopsy was done, radium directly implanted into the growth, a proceeding at the time thought by many to be tantamount to destroying the patient. The implantation was followed by heavy *x*-radiation at intervals for months. The patient is well at the present writing. The case is reported elsewhere. All other cases of osteogenic sarcoma have been treated either in conjunction with surgery or the treatment started late in the disease, and all the patients of whom we have knowledge are dead.

It is possible that in some instances the reputed cures of Ewing's tumor by irradiation have been examples of osteogenic sarcoma. Color is lent to this belief by the fact that favorable irradiation response is often the basis for diagnosis of Ewing's tumor and, hence, biopsy is omitted.

None of the cases of metastatic tumor of the skeleton which we have treated has had a long survival. However, enough relief of symptoms has been contributed to make this procedure well worth while.

The first requirement for the successful treatment of a primary tumor of the skeleton is that all the tumor should be excised or all irradiated to secure a favorable result. Therein lies the explanation of all failures of treatment whether surgical or by irradiation. If the surgeon excises all of the disease the patient will be well. If the correct amount of *x*-ray be distributed throughout the tumor the patient will also get well. The fact that a tumor is secondary in one location, that is, metastatic, means that it probably has other undiscoverable extensions in other locations which it is simply impossible to recognize and to irradiate or remove. This is the chief reason for not irradiating secondary tumor of the skeleton in the absence of symptoms.

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CHARACTERISTICS OF ADEQUATE ELECTROSURGICAL CURRENT*

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INTRODUCTORY

ELECTROSURGERY has reached its present stage of development almost entirely by an empiricism balancing between manufacturing agility on one side and clinical skepticism and daring on the other. Recent protest against claims of unstandardized and inadequate machines, comparable to patent medicines in lack of necessary available data concerning their physical characteristics and construction, has warrantably been made.[24]

Judging the subject too technical (or "super-scientific," as labelled by one council) for surgical inquiry, the medical profession, perforce, has had to be content to accept isolated and uncontrolled results as ultimate. Part of this lack of comparative criticism has been due to diffidence in the presence of a new technique, and part to lack of availability of varied apparatus and to lack of published material for wider comparison of performance. Beyond the initial impulse credited to Stoye by Maupin[14] and the pioneering close cooperation between Wyeth and a manufacturing firm's technical advisor, and between Bovie and the surgical staff of the Huntington (and allied) Hospitals, surgery, as far as recorded, has done little save to accept and try what has been presented as available. This paper records a series of data for several prominent American machines, and also attempts, through study of a flexible experimental construction, to rout some widespread misconceptions.

The electrosurgical unit, as utilized at present in major surgery, furnishes both coagulating and cutting currents. Recent wide adoption of modern devices has followed indeed upon the development of the latter, truly surgical, current. Coagulating currents, however, have long been known, and various modalities of high-frequency oscillations have been used in the treatment of both surgical and non-surgical conditions for almost half a century.

In 1881 Morton described before the New York Academy of Medicine the production and effects of induced static current.[16,17] In 1882, the physicist d'Arsonval did the same for d.c. low-frequency sine waves of various forms,[1] and nine years later reported similarly on alternating currents of frequency from two thousand to two billion per second;[2] two years later he reported heating effects in an electrostatic field within a giant helix;[3] meanwhile Riviere had used d'Arsonval currents for fulguration of intractable ulcers.[21] In 1907 Nagelschmidt reported on the indications and use of low-density high-frequency currents since known and utilized as diathermy.[19] In 1909 both Pozzi[20] and Doyen[9] detailed the use and technique of d'Arsonval currents for destruction of neoplastic tissue, and there began the dizzying differentiation and futile nomination of varieties of electrocoagulation, of which Clark's desiccation[7] alone is warranted clinically as a definite entity. Though Stoye's cutting current was created on a spark-gap machine producing damped waves (Fig. 1), gap construction for a time gave way to thermionic tubes (radio then being high novelty) and the peculiar idea of special physiologic qualities inhering in undamped cutting waves became propagated. In certain classes of work endothermic currents have such manifest advantages for dissection and hemostasis, over older methods that enveloping their

* Submitted for publication February 16, 1932.

modus operandi with hesitant and substantially unreal mystery, in the face of already known facts, can only be deplored. have so far been unable to find substantiating data for the claim for Germany in a search of the literature.

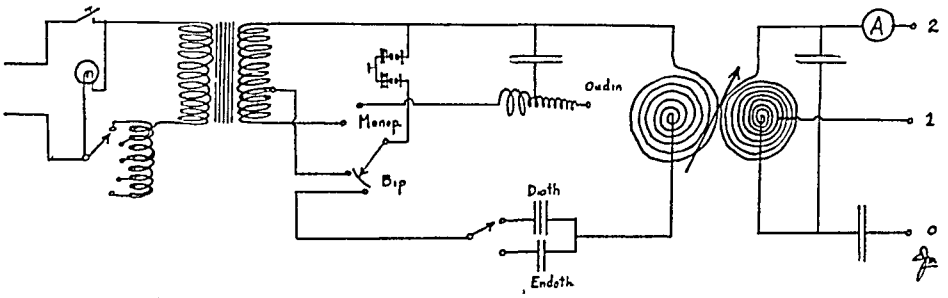


FIG. 1. Diagram of Stoye's clinically successful high frequency damped wave spark-gap endotherm.

The recorded early history of cutting currents is at present singularly obscure, and is contained only fragmentarily in footnotes and unearthed in chance discussions. In 1904, a Dr. Cohen unsuccessfully attempted a surgical operation at the Moabit Hospital, Berlin, with a damped cutting current machine devised by deForest. In 1907 deForest obtained his epochal patent on the triode tube. The following year he asked Weil and Sternberger at the Loomis Laboratory (Bellevue Hospital, New York) to undertake a few experimental operations with a cutting current machine he had devised utilizing the new tube; operations were successfully carried through on dogs. The device was also subsequently used there by Edwin Beer,[4] but deForest "never produced a machine which we could use" clinically. As already stated, Maupin attributes to Mr. K. Stoye, an electrical engineer, the production of the first successful cutting current, in 1918, presumably in Germany; I have been unable to confirm this in the literature or in personal communication with Stoye. Several American commercial concerns devised cutting current machines (1919–1923) whose results (because of unreliability, inexperience, burns, secondary healing, etc.) were such that widespread clinical adoption did not occur. It has been stated that in 1923 first applications of triode tubes to cutting were made in Germany, but the Loomis Laboratory machine antedates this by fifteen years. I

In 1923 George A. Wyeth became interested in working with high-frequency currents in the old Loomis Laboratory and, in conjunction with the research department of an eastern electromedical concern, devised and in May, 1924 described[27] a successful* triode undamped cutting current machine; it could also deliver selectively to the same electrode a d'Arsonval coagulating current. Stoye, who withdrew from the eastern concern, independently devised in 1924 the first successful† damped cutting current machine. Both Wyeth's and Stoye's machines were exhibited at the Atlantic City A.M.A. Convention of May, 1925. Of the subsequent flood of somewhat crude damped cutting machines (outgrowths of the old diathermic devices), that of a midwestern concern was persistently improved by the biophysicist Bovie, and, combined with a coarser gap current optionally deliverable to the same electrode, has been the popular van of electrosurgery's spread in this, and some foreign, countries.

To Wyeth, therefore, belongs credit for the first clinically successful undamped current endotherm, and to Stoye for the almost simultaneous, and first clinically successful, damped current endotherm.‡

*Two tube self-rectification.

† Spark gap firing for 85–90 per cent of each half-wave of the alternating cycle.

‡ "Endothermy," despite objections, is a suitable and useful term[26] to distinguish surgical high-frequency currents from medical high-frequency currents.

Electrosurgical (and diathermic) high-frequency currents at present range from about 200,000 oscillations per second to 4,000,000; neither physiology, physics, nor mathematics affords present ground for suspicion of unusual or cryptic effects within this frequency band. In the upper ranges of the band, and in frequencies above it, conduction delivery of current, necessary at present in electrosurgery,

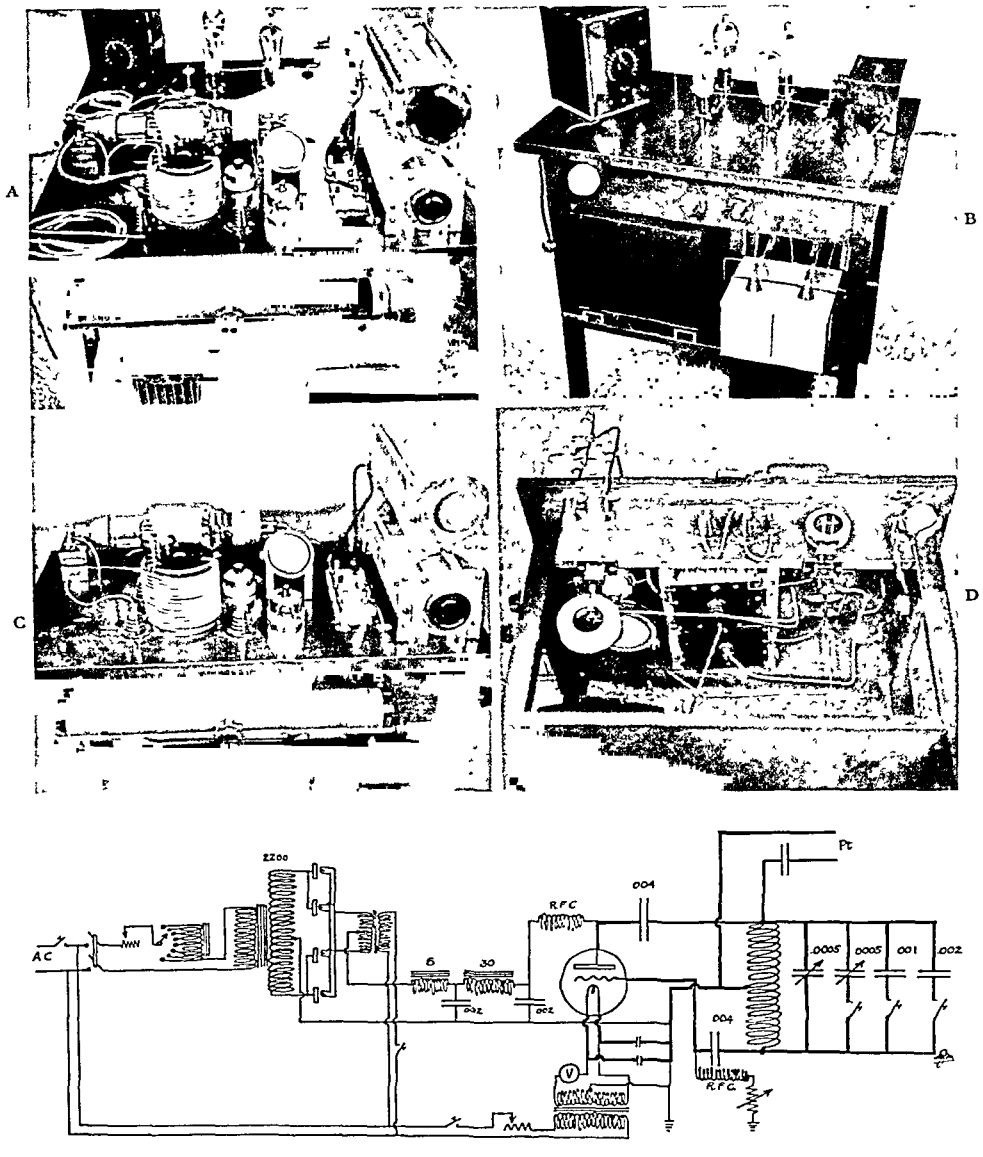


Fig. 2. Experimental unit constructed for this investigation. A. Assembled unit. B. Power table, where commercial alternating current at 110 v. was converted into direct current at 2200 v., and input to radio-frequency table controlled. C. Radio-frequency table, where direct current power was converted into oscillations ranging from 270,000 to 4,600,000 per second; variable condensers on right; radio-frequency coil, and triode tube, on left; sliding resistance for power, and variable resistances for grid leaks, are on sides of table. Knife switches key-in tuning condensers; turn switches control low-frequency power input, high-frequency power output and filament transformers; sliding resistance is supplemented by selective reactance shown on power table. D. Under side of radio-frequency table, showing fixed tuning condensers, protective condensers, and radio-frequency choke coils. E. Wiring diagram of experimental unit.

becomes increasingly difficult due to standing waves and power losses in cables of clinically necessary length (7–9 feet).

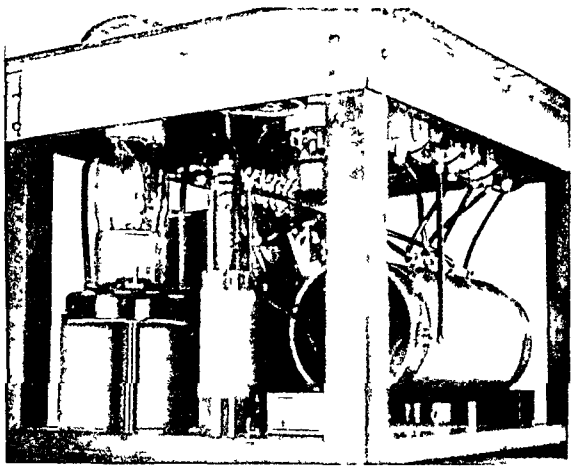


FIG. 3. A. Commercial triode machine B, showing one of power tubes, low-frequency transformer, resistances, choke coil, fixed tuning condensers and radio-frequency coil with taps and protective condensers.

Christie and Loomis,[6] however, demolished this by production of new evidence and by reinterpretation of Schereschewsky's results on a simpler basis of non-specific heat production. Hosmer[11] found in the lower reaches of this band some apparent selective heating effects of different frequencies upon various concentrations of saline solution. Woodbury[25] reported that Whitney, using about 50,000,000 frequency and about 200 times the power input utilized by Schereschewsky (7.5 watts), uncomfortably raised the temperature of nearby workers, cooked organic substances on neighboring antennae, and heated copper bars sufficiently to burn the hand when picked up; Bell and Ferguson[5] however reported that prolonged exposure to super-high frequency radio current under service conditions was substantially negligible, though describing an interesting series of

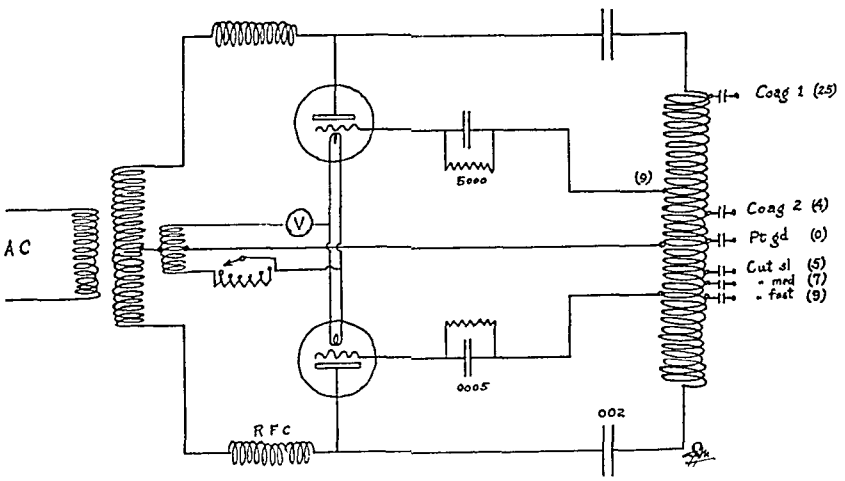


FIG. 3. B. Wiring diagram of machine B.

Electrostatic "conduction" however, involving niceties of resonant tuning (and stability and balance), has been studied by Schereschewsky[22] from 7,000,000 to 135,000,000 oscillations per second. Schereschewsky[23] in 1926 and 1928 believed he found a specific lethal frequency band ranging from 18,300,000 to 68,000,000, and, basing his interpretation on Lamb's mathematical work[13] for properties of elastic spheres, postulated for this band a form of destructive electromechanical vibration in cells of diameters from 7 to 96 microns. subjective and sensory phenomena derivable under special conditions. Beyond those frequency bands lie zones for heat; for infra-red, visible, and ultraviolet light; and for radioactive (*x*-ray and radium) oscillations.

Electrosurgery (endothermy) utilizes the production of intense local heat within the tissues; the intensity varies with current density. If the intensity is less than destructive, the current is conveniently called diathermic. In order to assure that heating effects only are produced, high-frequency

currents are used. One electrical stimulation per second of nerve or muscle causes an individual twitch in muscle; at 15 per second response is clonic; at 40 the muscle is in tetany and nerve trunk stimulation painful; above 2500 pain begins to diminish, but neuromuscular response remains maximal until 5000 and then decreases to 10,000; responses, other than development of heat, have vanished at 15,000. Modern electrothermic devices use frequencies from 80,000 to 4,000,000 and at such frequencies large amounts of electrical energy may be passed safely through the body without eliciting other than heating effects. This at present is the all-important and sole utility of high-frequency current as such. Cutting and coagulation have no fundamental bearing on frequency, oscillations, or wave form, contrary to current conception, and electrosurgical high-frequency is employed solely to assure the current's safe passage through the living body.

Electrical production of heat is dependent on quantity of current and amount of electrical resistance; in general it can be said that resistance decreases as frequency increases. If the resistance is encountered in the instrument through which the current is applied the instrument becomes hot, as in the electrocautery, where the current encounters resistance in the special wire tip of poor conduction properties, causing it to glow white hot (here the low-frequency current direct from lighting circuits is prevented from producing bodily harm by passage through a transformer, increasing amperage, but decreasing voltage below

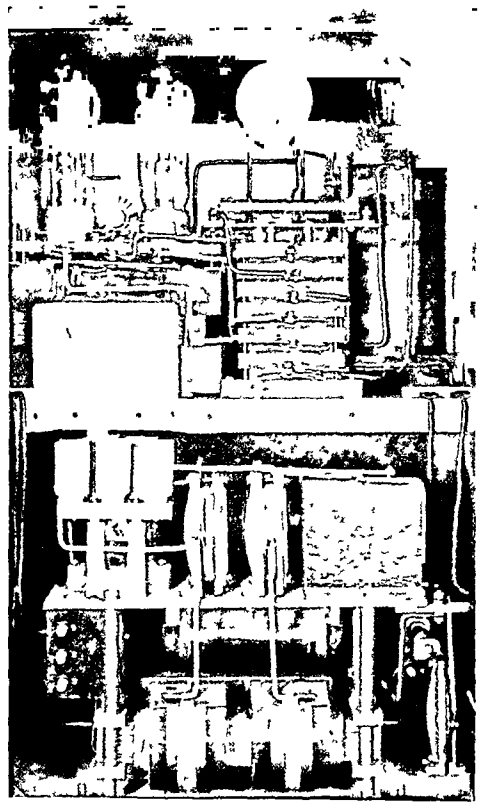


Fig. 4. A. Commercial triode and gap machine C. Cutting unit (triode) above and coagulating unit (gap) below; triode power tubes sit above low tension transformer and reactance; grid leak resistance is above bank of condensers, and neon lamp above radio-frequency coil and variocoupler. In lower unit vaned spark gaps, condensers, and Oudin coil sit above transformer and reactance.

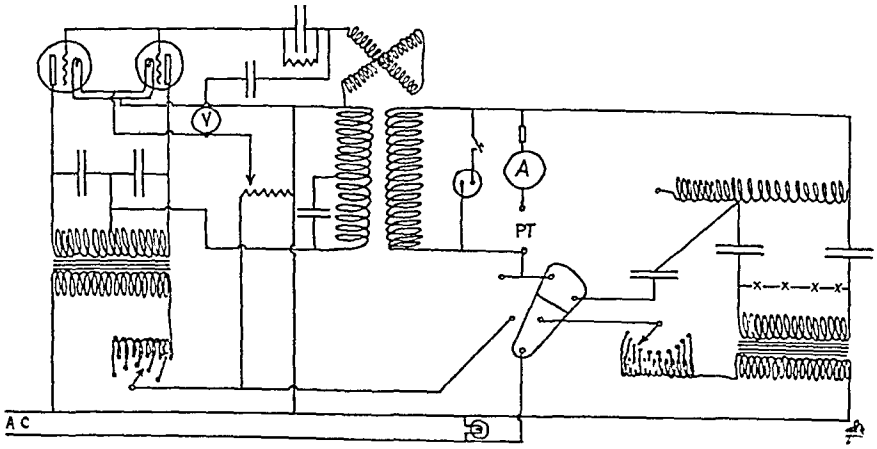


Fig. 4. B. Wiring diagram of machine C.

physiological stimulation range, the current's force being insufficient to produce direct physiological response). If the electrode tip be a good conductor, as in electrosurgical devices, the greater resistance is encountered within the tissues, and the heat is produced within the latter. In both, though the current is not painful, the heat is, and sensitive structures therefore must antecedently be anesthetised in a conscious subject.

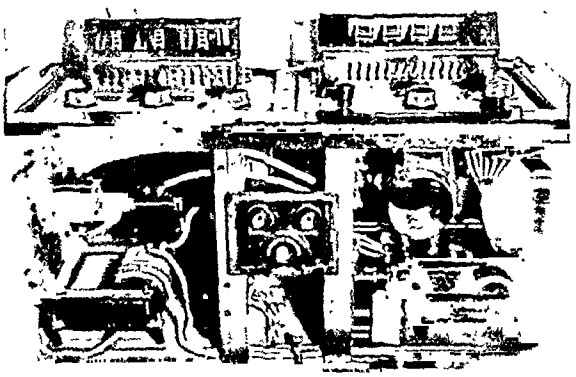


Fig. 5. A. Commercial gap machine D, cutting unit to left and coagulating unit to right. Micrometer spark gaps and dials above; transformers, coils, reactance, variocoupler and condensers below.

What effect the heat has is dependent on its intensity, and this in turn is electrically dependent on current density. A proper volume of current passing through the body between large (say, 8 inches square) electrodes is of low density, mildly warming, and not destructive, as in diathermy.[12] The same volume of current passed between a large and a small (say ¼ inch square) electrode produces a higher density at the smaller electrode, and coagulation of tissue ensues at the small electrode. That same volume of current passed between a broad electrode and a needle electrode

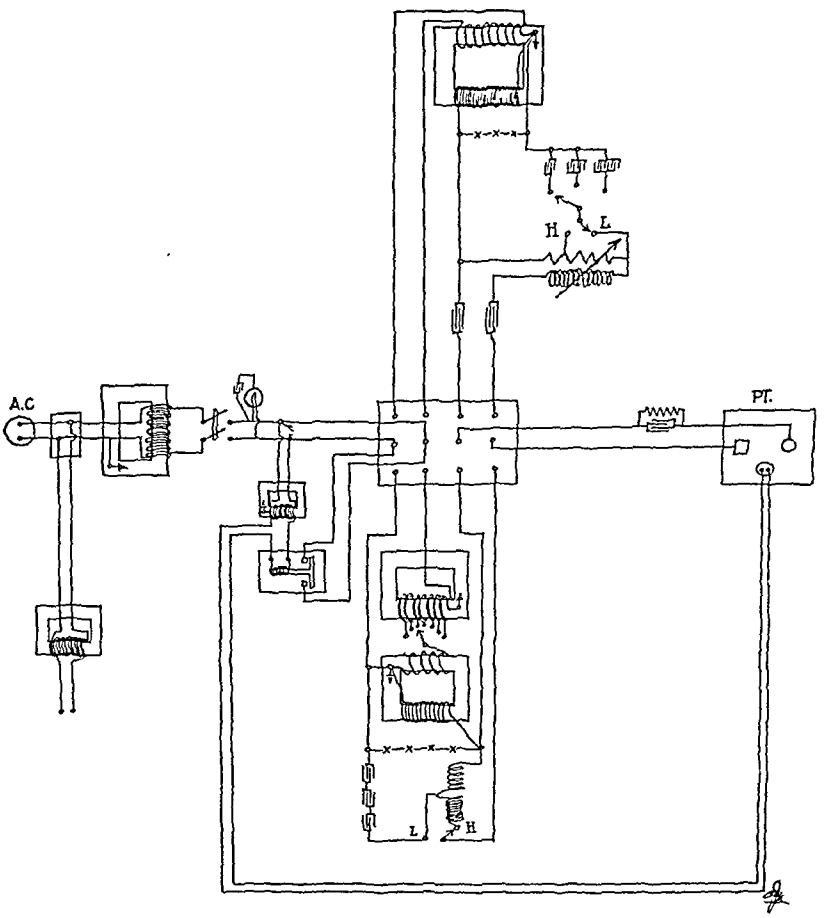


Fig. 5. B. Wiring diagram of machine D.

causes the intense local destruction analogous to a clean surgical incision. Obviously other factors also enter somewhat into the degree of heat developed in actual surgical work: the rate at which the small electrode moves, its size, the depth to which it is embedded, the degree of vascularization of the part, the character (degree of relative resistance) of the tissue. It is in evaluation and control of these latter as well as in the former that the empiric art of electrosurgery lies.

EXPERIMENTAL

Undamped oscillations have been considered as necessary to elicit cutting effects.

A. Experimental Unit (Fig. 2). *Construction:* In order to obtain as smooth (faradic-free) an experimental current as

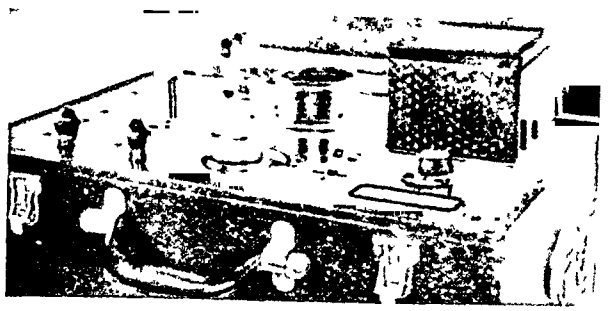


Fig 6. A. Commercial gap machine E. Spark gaps encased on right; variocoupler and reactance in foreground.

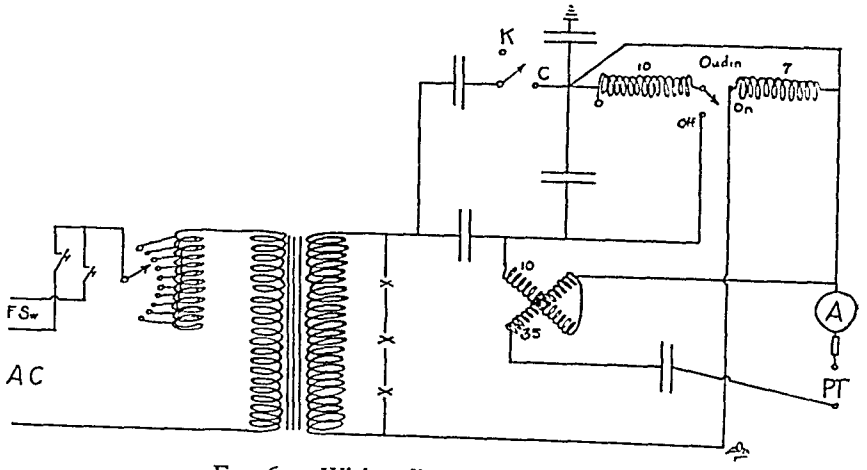


Fig. 6. B. Wiring diagram of machine E.

As a matter of fact an electrode delivery of 6 to 30 watts in undamped (triode) oscillations produces excellent cutting, while 20 to 75 are usually necessary with damped (spark gap) oscillations; the reasons for this have been considered elsewhere.[15] Since the efficiency of most electrosurgical machines of whatever type averages from 10 per cent to 30 per cent, less ruggedness and greater facility of construction has until recently been more possible with the former type of cutting apparatus. For experimental work, in addition, a proper triode set-up allows maximum flexibility in sharp control both of frequency and of amplitude of oscillation.

theoretically possible, filtered direct current was used to apply voltage to the tank circuit carrying radio frequency oscillations; a Hartley type radio-frequency circuit was chosen because of its reliability of performance. Interchangeable radio-frequency coils of large flat-wound copper wire were balanced against an ascending series of variable and fixed condensers to obtain oscillations ranging from 250,000 to 4,600,000; amplitude was flexibly controlled by a series sliding resistance and a 7-point reactance inserted in the power primary circuit, sufficiently strong at maximum to obliterate entirely any flow of current into the tank circuit.

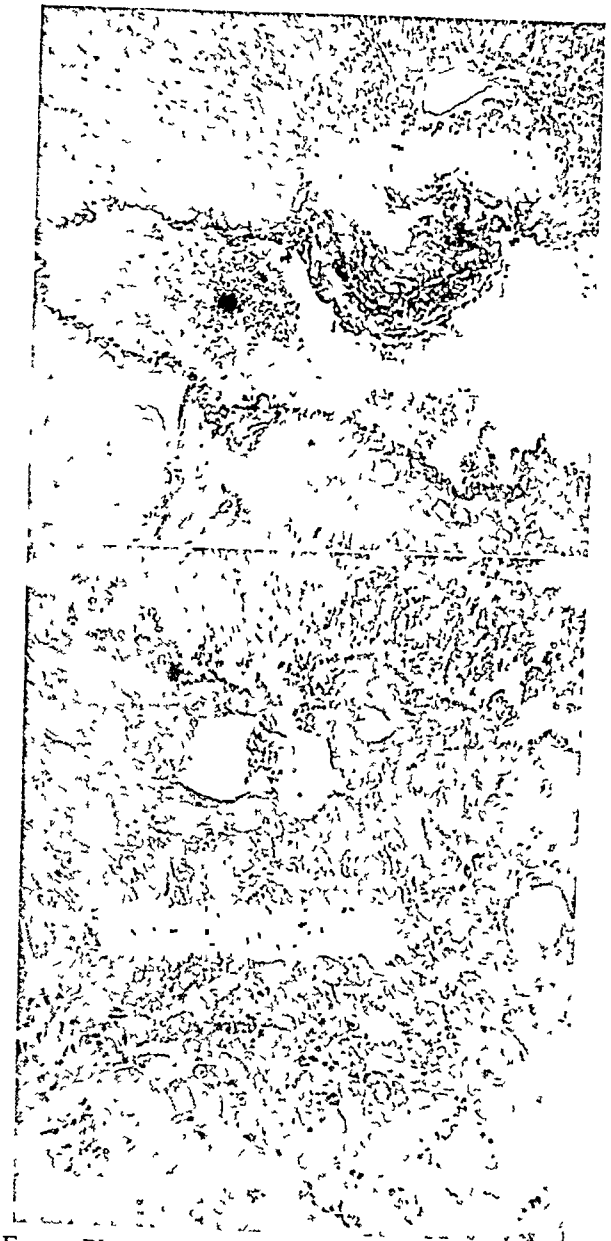


FIG. 7. Photomicrograph of incision in cat liver, made with 220 v. current at 60 cycles per second, electrode 0.013" diameter, 110 watts. There is 80μ of coagulation necrosis, with ground-glass cytoplasm and pyknotic nuclei, sparse carbonization of epithelium at surface of lip of cut for 3–6μ, and none in depths, there is also 15–20μ of coagulated amorphous serum under epithelium at surface for 150μ to each side. Blood in capillaries in coagulated area stains like hematin; some rare hemosiderin granules and clumps in lumen of incision; erythrocytes normal beyond 80μ. Some interstitial (30–50μ) steam vacuolization in coagulated area, especially at tip of needle. Cells appear normal beyond coagulated zone. H. & E., × 30 and 150.

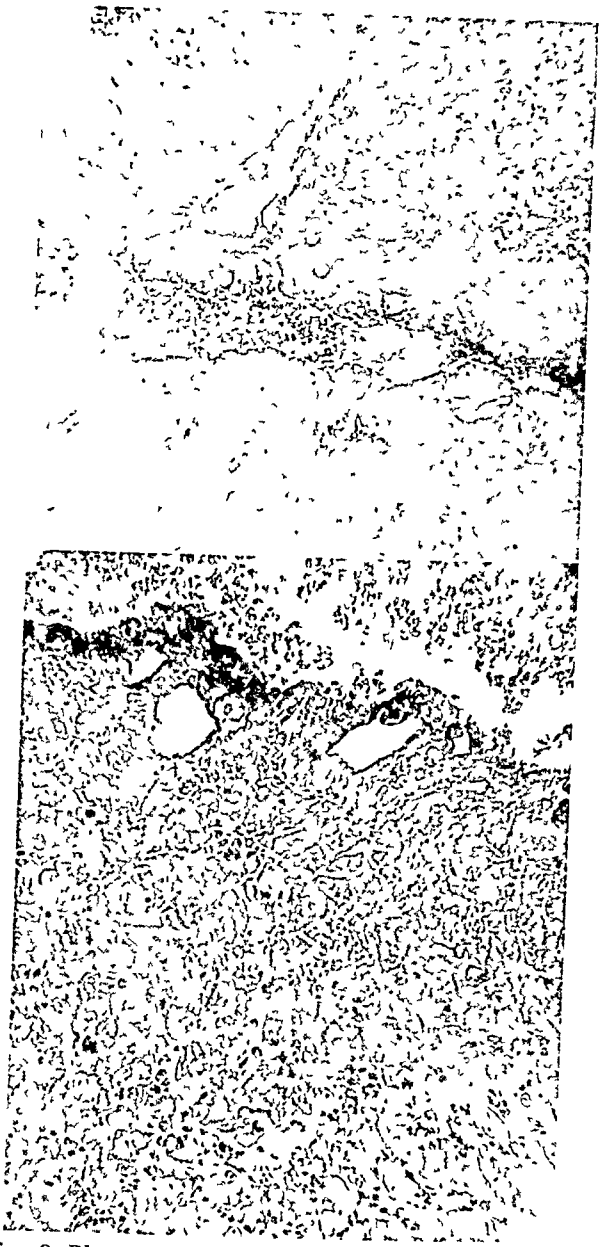


FIG. 8. Photomicrograph of incision in cat liver, made with 240 v. direct current, electrode negative and diameter 0.013", about 60 watts. A 3–5μ zone of carbonization, 30μ of coarsely reticulated nucleusless non-staining coagulum; 65μ classic coagulation necrosis, and unaltered cells beyond this; steam vacuolization minor. Cat used in this and Figure 7, was fat and had long been in laboratory; concerning evident non-specific hydropic changes in liver cells, see Ref. 10, p. 131. H. & E., × 30 and 150.

Sixty-cycle current at 110 v. was fed to the primaries of three transformers: (1) Acme 670 supplying 2.5 v. and 5 amp. to each of the 4 mercury vapor radiotron rectifier tubes, (2) G. E. UP 1016 supplying 11 v. and 15 amp. to the filament of the power oscillator tube, (3) and Acme 5181 drawing 10 amp. and supplying ultimately 2200–3000 v. to the oscillator tube plate. The current to the last transformer was passed through a porcelain core sliding resistance in series with a specially wound 7-point reactance (choke coil), these affording control of amplitude. Four UX 866 radiotrons were used to rectify the 2200 v. alternating current from the power secondary, and the resultant unidirectional current smoothed in a filter unit composed of Acme

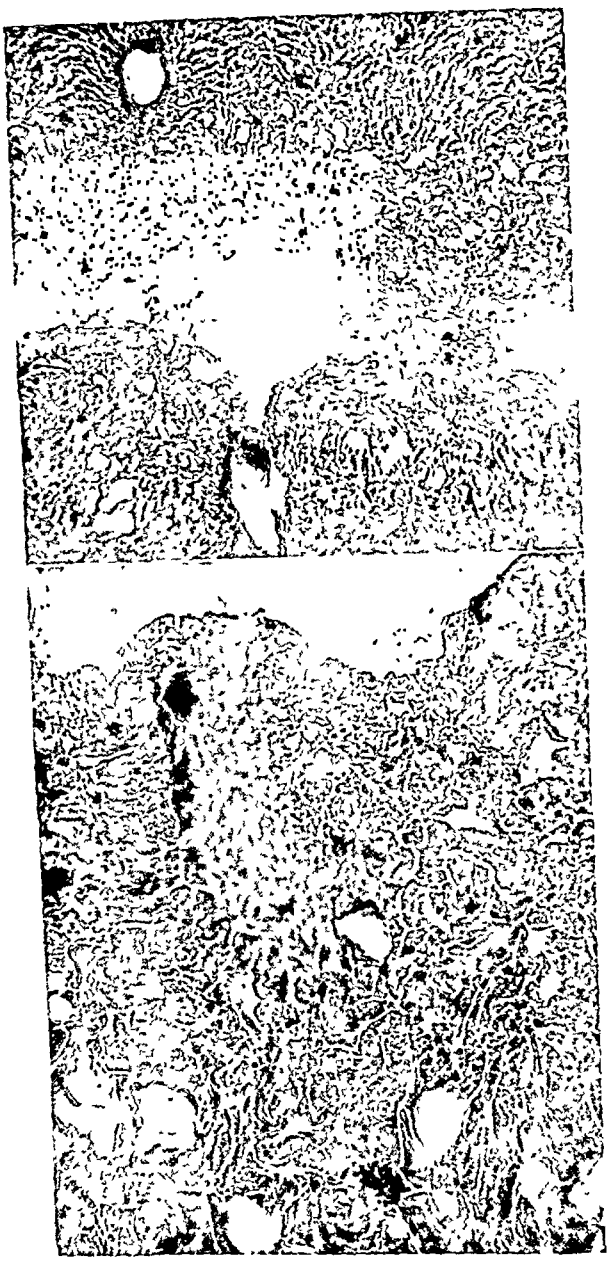


FIG. 9. Photomicrograph of incision in cat liver, made with 0.013" electrode at 4,600,000 oscillations per second and utilizing 45 watts (Table 1, line 18; and Table 6, line A, 9). Total zone of alteration about 250 microns. H. & E.. × 30 and 150.



FIG. 10. Photomicrograph of incision in cat liver, made with 0.013" electrode at 1,060,000 oscillations per second and utilizing 30 watts (Table VIII, line A, 5 and Table 1, line 13). Total zone of alteration about 200 microns. H. & E., × 30 and 150.

680 6-henry choke coil and a 674 30-henry choke, with two intercalated Tobe 3320 2-microfarad transformer condensers. The radio-frequency honeycomb choke coil between plate and filter, and, through the grid leak, by a similar coil interposed before the grounded

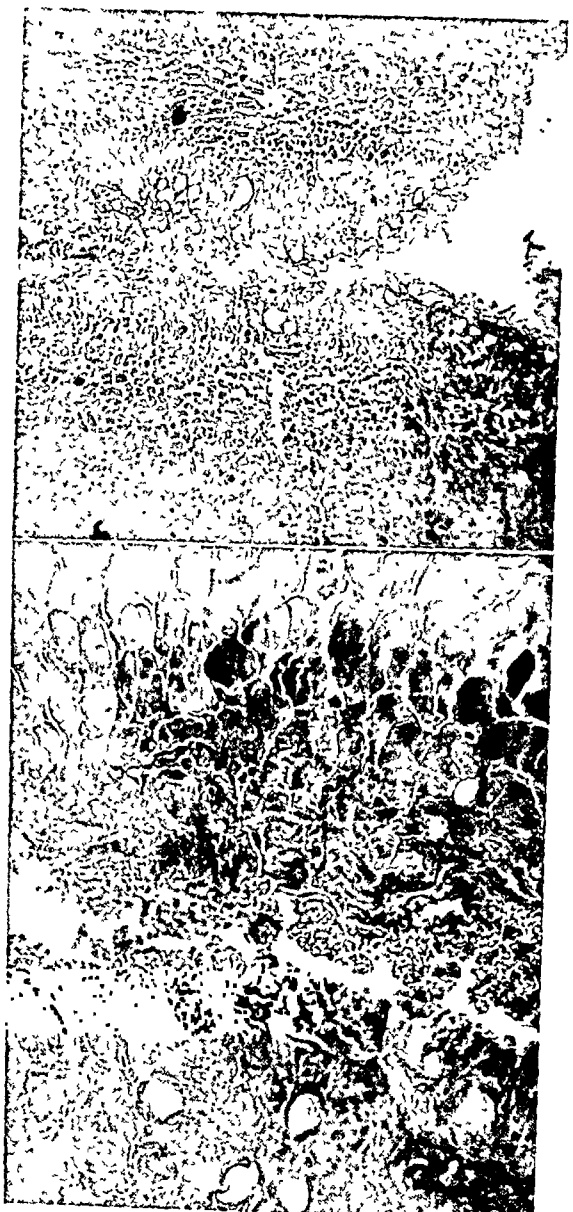


FIG. 11. Photomicrograph of incision in cat liver, made with 0.013" electrode at 3,000,000 oscillations per second and utilizing 100 watts (Table II, line 14 and Table VIII, line B, 1). Total zone of alteration about 60 microns. H. & E., × 30 and 150.



FIG. 12. Photomicrograph of incision in cat liver, made with 0.013" electrode at 3,000,000 oscillations per second and about 70 watts (Table IV, line 2 and Table VIII, line D, 6). Total zone of alteration about 300 microns. H. & E., × 30 and 150.

direct current was then fed to the plate of a 250 watt G. E. UV 204-A, type 251, triode power tube, incorporated in the Hartley circuit; escape of high-frequency oscillations through supply lines was blocked by a 150-turn variable resistance; grounding was accomplished readily through the indifferent wire of the supply lines. The alternating current voltage of the filament was smoothly held constant during operation by grounding the

midpoint of the secondary and each side of the filament, the latter through small 0.0005 by-pass condensers blocking escape electrode. This conduction delivery was thus adequately safeguarded and avoided the clinically prohibitory tuning of another inductively

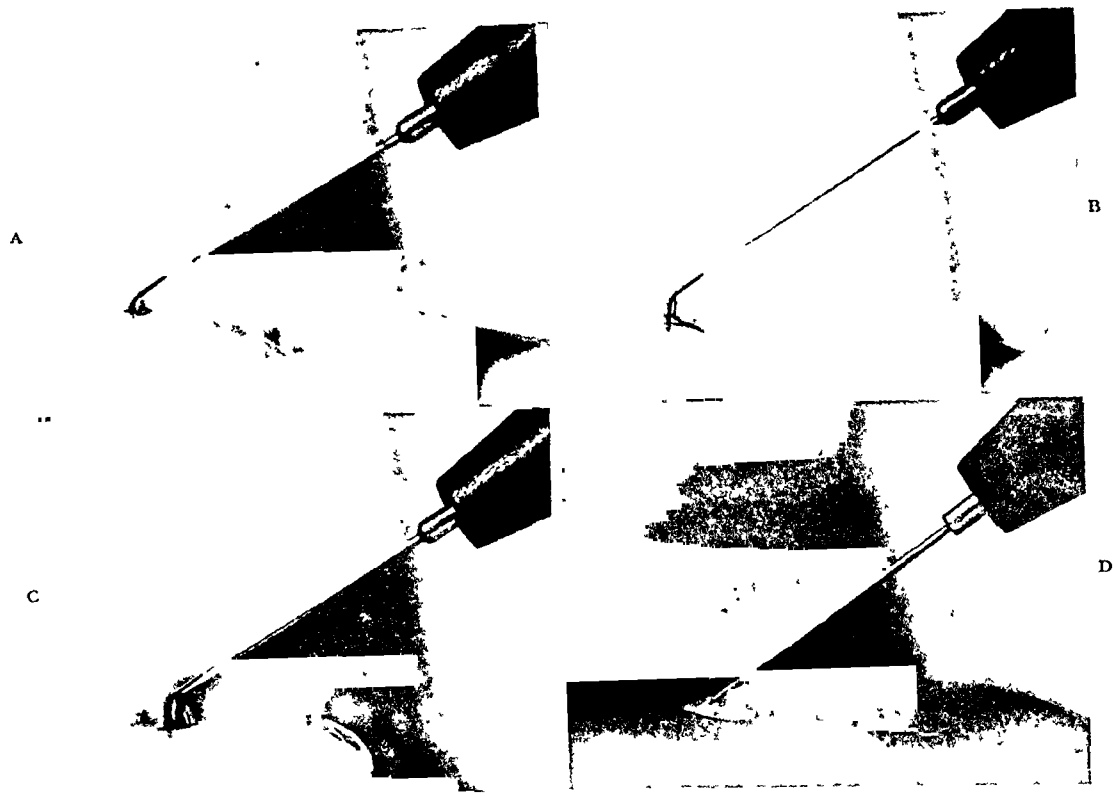


FIG. 13. Instantaneous photographs (1/200 second) of discharges produced by coagulating type currents. A, B, and C are from commercial machine D, low voltage medium dehydration, high voltage medium dehydration, and low voltage heavy dehydration. D is from commercial machine B, low voltage.

of the low-frequencies necessary for filament incandescence. The grid connection of the interchangeable radio-frequency tuning coils was fixed; the ground and plate leads were of flexible heavy copper tape terminating in broad clips for selective attachment to the coil; the coils, 5 inches in diameter, were either of copper tubing or of flat-wound heavy copper strip, 15, 30, and 60 turns. The 4 tuning condensers were 2 National TMU 500 variables of 0.5 microfarad capacity and 2 R.C.A.'s (3395 and 1082) of 1 and 2 microfarad capacity respectively, so arranged that they could be successively keyed into the tuning circuit. The patient was protected from low-frequency power currents and from possible flash-over surges in the oscillator by two 0.004 Dubilier condensers interposed between plate and coil, and grid leak and coil. Additional safety was insured, in case of stress breakdown of either of these latter, by a third Dubilier inserted in the cable to the active coupled circuit, with its rare but troublesome fading in emergencies.

Mensuration: Filament voltages, and power table output in amperes and volts, were measured with customary meters. Power output from the radio-frequency table was measured both at the table terminals and again within a few inches of the electrode tips; in the appended tables only the latter values are given;* for measurements here Weston Electric radio-frequency ammeters ranging from 100 ma. to 20 amp. were used; and Rawson electrostatic voltmeters ranging from 100 to 2500

* What goes into a machine, what it delivers at its terminals, and what is delivered at the electrode tips, are entirely distinct things, particularly in high frequency apparatus. It is with the third one of these that the surgeon is concerned, and into which he should, and this article does, inquire.

v., higher voltages being measured on a special ball-electrode micrometer spark gap. The complete accuracy of the foregoing measuring devices, at upper ranges of frequencies investigated, may be doubted. Wave length and frequency were determined with a General Radio precision wavemeter, Type 224, having range from 70 to 24,000 meters; frequency was determined both at coils of machines and in final reaches of the cable supplying the active electrode. Electrode diameters were measured with a screw micrometer. Measurements of resistance were made on a direct current potentiometer. Time for incisions was usually estimated with a stop watch.

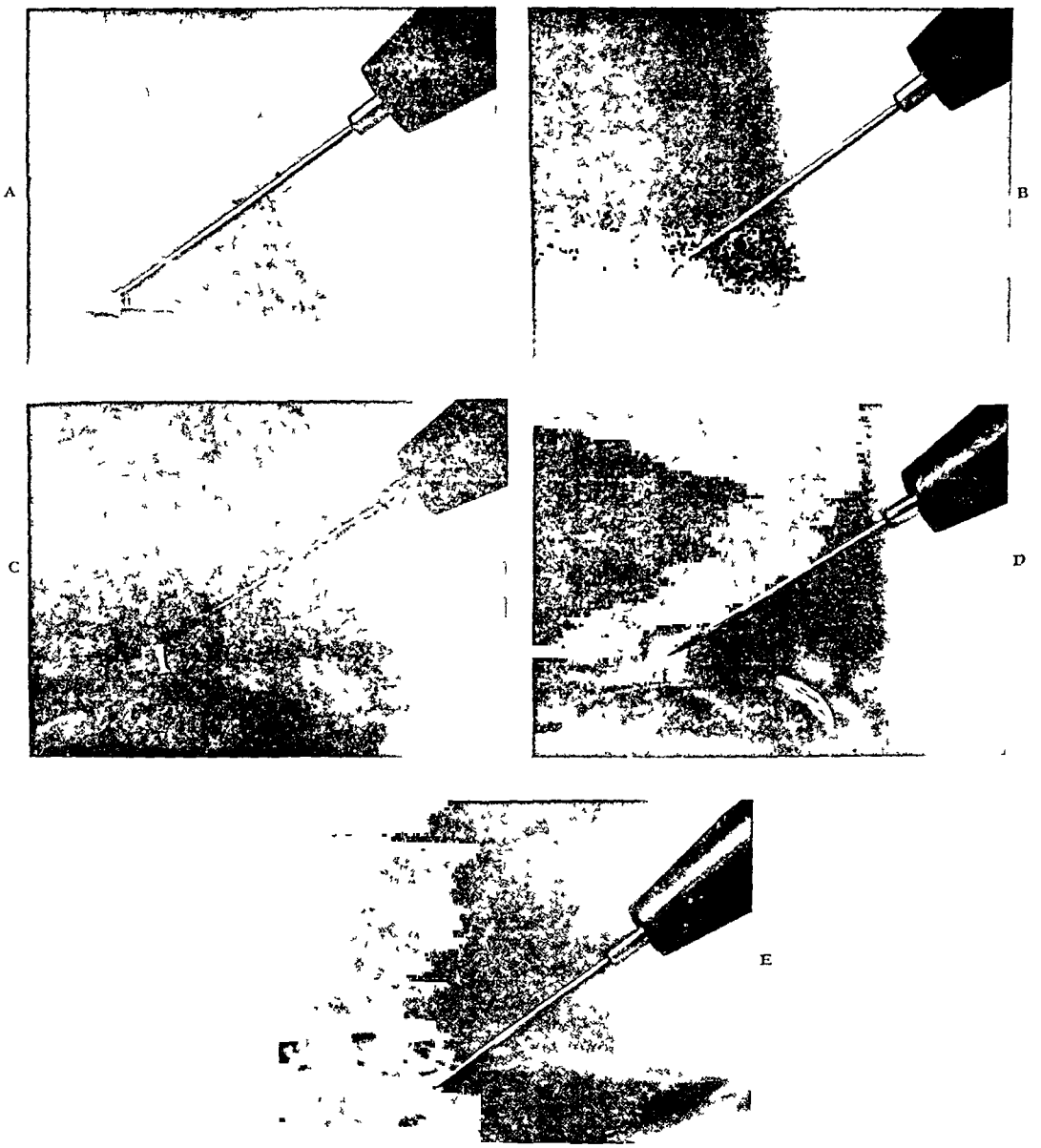


FIG. 14. Instantaneous photographs of discharges produced by cutting currents. A and B from experimental unit, C and D from commercial machine D, and E from commercial machine E.

Fresh or living liver (4700–9000 ohms) was used uniformly for making incisions, because of the ease with which zones of alteration may be estimated in gross.

These estimations are relative, but have been checked histologically personally many times in the past as well as frequently during the progress of this present work; only in gaping and everted incisions made with high voltage, producing flame burns, is there persistent tendency to overestimate somewhat in gross the extent of microscopic alteration. Tissues were uniformly fixed immediately in Zenker's solution, embedded in paraffin, stained with eosin-methylene blue or with hematoxylin and eosin. Histologic zones of penetration were measured with a calibrated ocular micrometer.

Results: The results are tabulated as follows (Table 1):

delivered to the active electrode; below this voltage cutting did not appear. The rate at which the incision could be made was a rough function of the delivered wattage; and the extent of lateral coagulation tended to parallel the delivered amperage, with an optimum mean of about 100 ma. Frequency had no appreciable bearing upon quality, rate or character of incision. Though more power apparently was impounded in the tank circuit here at peak in higher frequencies, only amounts of power roughly homologous to those at lower frequency were deliverable (or desirable) at the electrodes.

When peak voltage fell below critical 200 only desiccation effects were possible.

Table 1

	Input (Watts)	Output					Incision 3 Mm. Deep and 10 Cm. Long		Remarks
		Voltage		(Ma.)		Watts			
		Peak*	Op.*	Peak	Op.	Op.	Time	Lat. Coag. μ	
270,000/sec. 1	101	1,200	450	55	300	135	0.3	700–900	Bad flame burn 2–6 mm.
2	95	1,050	275	50	250	68	0.7	200–400	Bad flame burn 1–2 mm.
3	84	800	275	45	160	44	1.0	200–400	Slight burn
4	41	500	225	30	80	18	3.5	100–300	Good incision
5	31	250	225	15	25	5.6	8.0	100–200	Good; very slow
6	9	50	25	±	±	?.02			Spt. desicc.
600,000/sec. 1	89	950	275	160	140	38	1.5	150–250	1–2 mm. flame
2	72	825	250	145	120	30	3.0	100–150	0.5 flame
3	42	650	225	110	80	18	4.5	100–150	All right
4	25	375	200	40	30	6	6.5	100–150	Good; slow
5	10	175	0	15	0	?o			Spt. desicc.
1,060,000/sec. 1	59	950	225	350	100	22.5?	3.5	200–500	0.5 burn
2	54	825	250	300	120	30	3.8	200–400	All right
3	48	650	225	260	110	24.7	4.0	150–300	All right
4	27	425	225	170	100	22.5	6.5	150–300	All right
5	18	250	225	100	55	12.4	17.0	50–100	All right
6	7	?25	25	20	20	0.5			Vspt. desicc.
4,600,000/sec. 1	103	800	150	900	300	45	2.5	100–200	Brilliant arc under tissue
2	74	550	125	650	300	37	3.0	100–200	High whine
3	33	440	150	500	250	37	5.5	50–150	High whine
4	14	300	200	310	200	40	12.0	50–100	
5	4.8	50	0	230	0	?o			Vspt. desicc.

* "Peak" readings, throughout these tables, are measurements of energy just before the active electrode is placed in contact with the tissue; "operative" readings are those obtained during the actual cutting or coagulation. Both were obtained by instruments within a few inches of the active electrode tip.

Comment: From Table 1 will be seen that good cutting occurred when 200–225 v. were

Optimum wattage for a good incision had a mean about 18–25, and when peak

voltage rose above 750 the operating voltage tended to rise above 225 volts (optimum) with resultant production of undesirable flame burns on the surface.

B. Commercial Triode Machine (Fig. 3). This recent compact machine, operating at a fixed frequency of 3,000,000, omits the unnecessary direct current for plate voltage, and applies stepped-up alternating current directly to the plates of two triode tubes acting as self-rectifiers and each using one-half of the low-frequency cycle. Amplitude of oscillation is primarily controlled by electron depletion within the tubes, through a 5-point tube filament rheostat; grades of cutting and of coagulation are further obtained by tapping the radio-frequency coil at varying distances from its center ground, each lead passing through a small protection condenser.

Construction: Sixty-cycle alternating current at 110 v. is fed to a primary coil of a transformer exciting two secondary coils, one, a step-down coil, supplies the 10 v. for the filaments through a 5-point rheostat, and the other, a step-up winding, supplies the thousand volts to be impressed on the plates of two G.E. UV 203-A tubes. Loss of radio-frequency oscillations into supply lines is prevented by radio-frequency choke coils of about 130 turns of cotton-covered wire (20 turns per centimeter) on a bakelite core 3.5 cm. diameter. Grid leaks are taken care of by 5000 Vitrohm resistances bridged across 0.0005 condensers. Oscillation frequency is determined by the grid tap (at 9 turns) on the r.f. coil and by the small 0.002 mfd. Dubilier mica condensers (Type 580). The r.f. coil itself is 49 turns of 1 mm. bus wire on a grooved 9 cm. hollow bakelite core, with 3 mm. space between turns, the coil being tapped at center for ground (body), at 5, 7, and 9 turns for slow, medium, and fast cutting, and at 4 and 25 turns for coagulation and desiccation; each tap is led through a protective 0.0005 mfd. condenser.

Results: The results are shown in Table II.

Comment: Here again the rate of incision paralleled the delivered wattage; the optimum however was between 40–80 watts. When the delivered voltage rose above 250, surface flaming occurred, marring the incision. The milliamperage delivered for satisfactory incisions ranged from 190–300.

During coagulation, peak and operating voltage, amperage, and wattage were identic. The voltage remained low, and when it reached critical 200, cutting effects appeared. With the appearance of cutting at critical voltage, peak and operating identic relation no longer held, as power was absorbed in persistent arc ionization.[15]

C. Commercial Triode and Gap Machine (Fig. 4). This excellent pioneer of generally utilizable clinical machines met squarely the necessities of its time by incorporating a flexible triode cutting outfit, and a versatile spark gap coagulating outfit in one cabinet, both independently feeding a single pair of electrodes. It however has a multiplicity of necessary controls, and requires what is generally considered bothersome tuning despite the fact that it operates at a substantially fixed frequency. Being inductively coupled to the patient, its delivery circuit sometimes fades annoyingly, although this clinical unreliability is exceedingly rare.

Construction: Sixty-cycle alternating current at 110 v. is led to a selector switch diverting it to either cutting or coagulating units. On the cutting side, it passes through a 5-point reactance (choke coil) feeding the high tension primary of a transformer whose secondary winding charges condensers and furnishes voltage to the triode plates. The low-tension current also lights the triode (two 50 watt 211-D Westinghouse) filaments, whose voltage is controlled to steady maximum efficiency through a rheostat thrown across the line. The charging of the condensers momentarily activates the primary of the r.f. transformer and inductively excites controlling oscillations in the neighboring variocoupler and its parallel condenser, these oscillations being transmitted to the grid. Frequency at about 300,000 is largely determined by the fixed tuning condenser across the plate end of the r.f. primary, while amplitude is controlled through the grid, though this circuit also secondarily affects frequency; this gives two controls of

amplitude: reactance and grid. The secondary of the r.f. transformer, together with conducting cables and patient, form a third circuit, and this is brought into resonance with the tank circuit by the variocoupler, the neon glower lamp being used to find maximum efficiency of power transmission.

When the selector switch is thrown to the coagulating unit, it again passes through a controlling reactance before reaching the high tension transformer; beyond this are four air-cooled micrometer spark gaps and two condensers, in the usual arrangement for production of damped oscillations;[18] the patient is further protected against electrical defects by intercalation of another condenser in one of the cables to the terminals. The excitation of the hot end of a Oudin coil (for desiccation) is controlled by a separate switch on the panel face.

Table II

	Output					Incision 3 Mm. Deep and 10 Cm. Long		Remarks
	Volts		Ma.		Watts	Time	Lat. Coag. μ	
	Peak	Op.	Peak	Op.	Op.			
Rheo.								
Slow cutting 1*	225	220	160	190	41.8	3.5	100–200	
2	225	220	190	235	51.7	2.5	100–200	
3	275	225	225	250	56.2	2.0	100–200	Sl. brown sear
4	275	225	225	300	67.5	1.5	100–200	
5	300	225	275	315	70.8	1.2	150–250	Initial flame
Med. cutting 1	225	215?	160	190	40.8	2.5	150–200	Clean beautiful current
2	250	220	200	250	55	2.0	150–200	
3	300	250	250	300	75	1.5	175–250	
4	325	250	290	350	87.5	1.2	175–200	Initial slight burn
5	380	250	310	400	100	1.0	175–200	Initial slight burn
Fast cutting 1	300	225	225	275	61	2.0	150–200	
2	350	225	275	310	69.7	1.5	150–200	
3	400	250	300	350	87.5	1.2	200–300	Flame burn each end
4	440	250	300	400	100	1.0	200–300	Flame burn each end 30–80μ
5	500	275	400	490	134.7	0.7	200–400	Flame burn each end 30–80μ
Coag. 1	100	100	±	±	0.1	0	0	0.5 mm. in 1 sec.†
2	150	150	50	50	7.5	0	0	1 mm. in 2 sec.
3	175	175	100	100	17.5	0	0	
4	200	200	150	125	25	4.5	150–200	1 mm. in 1 sec.
5	225	200	200	150	30	4.5	200–250	Burn hole about needle; 1 mm. coagulation beyond
Desicc. 1	650	50	350	0	0.05?	3.5	200–500	Gapes
2	775	150	450	0	0.05?	3.5	200–400	Gapes
3	825	150	500	75	12	4.5	300–500	Gapes
4	800	200	500	75	15	4.5	500–600	Gapes
5	900	200	550	75	15	2.5	500–600	Gapes

* Filament voltages, rheostat stop No. 1, 7.5 v.; No. 2, 8.0; No. 3, 8.5; No. 4, 9.5; No. 5, 10.2 volts.

† Coagulation was uniformly tested throughout the investigation by inserting needle electrode 1 cm. into tissue, and noting time to produce maximum effect.

Results: The results are given in Table III.

Comment: Triode unit: Adequate cutting effects were obtained with a delivery of 15–25 watts. When the delivered voltage rose above 225, flame burns about the surface of the incision detracted from

TABLE III

Frequency	Output					Incision 3 Mm. Deep and 10 Cm. Long		Remarks
	Volts		Ma.		Watts	Time	Coag. μ	
	Peak	Op.	Peak	Op.	Op.			
a. Triode cutting panel:								
300,000 (Variom. 7)								
1	150	150	0	40	6	6″	240–300	Feeble cut
2	200	200	0	100	20	3½	200–300	Fair cut
3	275	225	0	100	22.5	2½	400–500	Good cut
4	350	225	0	160	36	2	100–200	Slight flame start
5	440	300	+	250	75	1.5	300–700?	More; side of incis. seared tan
312,000 (var. 3)								
1	150	150	0	70	10.5	5	150–200	
2	225	175	0	100	17.5	3½	200–400	Superb cutting
3	300	225	0	110	24.7	2½	300–400	
4	375	250	0	150	37.5	2	200–300	
5	440	300	+	230	69	1.5	300–400	Slight flame burn
292,000 (var. 0)								
1	0	50	0	20	1.0	9	100–200	Very poor cut
2	175	150	0	80	12.0	4.5	200–400	
3	250	225	0	110	24.7	3.0	200–300	
4	300	225	0	140	27.5	2.0	200–300	
5	400	300	+	220	66.0	1.5	200–400	Slight flame burn
b. Triode coagulating panel:								
272,000								Coagulation:
(var.-3) 1	...	...	..	..		Vspt.	Desicc.	1.5 mm. in 4 min.
2	...	...	..	..		Vspt.		2.0 4
220,000 3	...	...	..	..		Vspt.		2.0 2½
(var.-7) 4	...	...	..	..		Vspt.		2.0 1½
5	200	175	0	45	10.5	6½	400–600	2.0 ½

c. Spark gap panel: Frequency: A broad band extending from 337,000 to 800,000, with its major output between 525,000–571,000; alteration of gap aperture does not appreciably affect distribution of frequency.

	Coagulation								Fulguration								
	Voltage		Ma.		Watts	T.	Change Diam. mm.	Crater Diam.	Voltage		Ma.		Watts	J*	D*	C*	D*
	Peak	Op.	Peak	Op.	Op.				Peak	Op.	Peak	Op.	Op.				
1	70	±	0	80	0.08	60″	8	0.1	0	75	0	20	1.5	1	Spray, without effect		
3	175	150	0	250	37.5	30	15	1.0	75	100	±	70	7.0	4	5	0.5	2.5
5	250	200	30	380?	76?	15	15	3–4	200	225	20	70	15.7	5.0	5	1	2.5
7	225	200	50	340	68	15	15	3–4	225	225	30	90	20.3	5	5	2	3
9	250	225	50	300	67.5	8	15	3–4	250	225	50	130	29.3	5	6.5	3	3.5
10	...	...	..			..	..	...	300	300	70	150	45	5	6.5	3	3.5

Table III (*Continued*)

d. Oudin current:
Frequency 1,363,000–1,430,000

	Voltage		Ma.		Watts Op.	J	D	C	P
	Peak	Op.	Peak	Op.					
1	400	400	±	±	?	5	Pract. nothing		
3	600	600	±	±	?	7	3	0	300
6	925	850	±	±	?	7	4	0	500
9	1000	850	±	±	?	7	4	0	500

* J: distance (mm.) intervening between electrode tip and tissue at time spark first jumped. D: diameter of superficial change produced in tissue (mm.). C: width of crater blasted in central part of coagulated area. P: depth of penetration of coagulation into tissue.

surgical desirability. Frequency low, and here, as in first section of Table I, a smaller recorded wattage produced more lateral coagulation than greater amounts at higher (Table II) frequency.

Gap unit: In coagulation a maximal radial penetration of 7.5 mm. could be attained with 35 watts; increased amperage only lowered the time for this penetration to occur; again, peak voltage above 250 produced charring. In fulguration a maximal spark of 5 mm. was obtained, at 225 v.; above this, only amperage showed consistent increase, and with it some minor increase in the zone of coagulation, at the expense of carbonization of the central zone. In desiccation (Oudin coil), the spark was 7 mm., dispersion and penetration far less; the amperage was immeasurably low and the voltage ranged from 500 to 1000.

D. Commercial Spark Gap Machine (Fig. 5). This steadily improved sturdy apparatus has been the outgrowth of one of the early primitive cutting machines and its reliable performance has contributed materially to the strong entrenchment of electrosurgery in this country.

Construction: Line supply of alternating current, 60-cycles, 110 v. and 10 amp. is introduced on the left of the diagram. A tap leading to an insulating transformer supplies current for the operator's headlight, and to the insulating transformer for the entire machine. This transformer, as well as all others beyond it is grounded through the supply wiring in order to keep the machine's oscillating potential symmetrically above and below zero potential. A main line switch, pilot lamp, auxiliary contact switch are interposed before the giant selector switch is reached.

The median four terminals of the selector furnish a low-frequency inlet, and an egress to the patient for the high-frequency current produced by either of the units depicted above and below it; it is arranged to afford contact with either marginal set of four terminals, throwing into circuit the upper (cutting) unit, or the lower (coagulating) unit, actual egress of the current into either, however, being controlled at a distance by the operator through the magnetic relay (foot or hand) switch.

In the cutting unit, the voltage is stepped up by a special transformer, and used to charge selectively one of three condensers of fixed capacity; the larger the condenser, the greater the initial amplitude of the damped wave, and the resultant dehydrating penetration in the tissue. Flexibility is further enhanced by introduction of a variometer with a selective low or high voltage winding on the outer (stator) drum, the inner drum being arranged to revolve at will to cut with increasing effectiveness the electromagnetic lines of force set up by the stator windings. Additional condensers are interposed on each line beyond the variometer in order to protect the patient from low-frequency jolt if a fault should develop in the dielectrics of the condensers of the generating circuit.

In the coagulating unit, current led into a reactance is tapped off at any one of 7-points and fed to a step-up transformer, beyond which are a coarser spark gap and three condensers in series, the latter arrangement being chosen for safety to reduce the pressure

TABLE IV

The fundamental frequency of the cutting unit is 3,000,000 though as the variometer is increased the frequency falls to 2,500,000; with light dehydration when varimeter is at maximum, at both low and high voltage, a considerable number of bands appear from 1,600,000 to 4,000,000; if gaps are widened at any time the frequency band is maximally broadened by 50,000–100,000 oscillations. The coagulating unit produces several close set frequencies; the main band (probably 60 per cent) is at 1,111,000, secondary bands are at 1,000,000 and 1,350,000; and a tertiary band at 1,200,000.

Cutting unit:

	Low Voltage							High Voltage						
	Volts		Ma.		Watts	Incision 3 Mm. Deep and 10 Cm. Long		Volts		Ma.		Watts	Incision 3 Mm. Deep and 10 Cm. Long†	
	Peak	Op.	Peak	Op.	Op.	T.	Coag. μ	Peak	Op.	Peak	Op.	Op.	T.	Coag. μ
Var. Light 0	0	0	0	0	0	...		0	0	0	0	0		
50	600	225	310	250	56	2	50– 100	1025	250	425	275	68.7	2	500–1000
100	875	250	350	275	68.7	0.5	200– 400	1025	250	400	400	100	1	200–1000
Med. 0	0	0	0	0	0	...		0	0	0	0	0		
50	450	225	200	350	78.7	2.5	100– 200	800	200	400	250	50	0.5	500– 800
100	600	250	250	300	75	0.5	500– 700	800	225	350	450	101.2	0.5	1000–1500 c burn
Heavy 0	0	0	00	0				0	0	0	0	0		
50	400	205	160	240	48	4.0	400– 600	400	200	100	75	15	0.5	500– 700 poor
100	700	250	200	600	150	0.5	700–1000	775	220	300	400	88	0.5	1000–2000 c burn

*Fulguration:**

	Low Voltage									High Voltage								
	Volts		Ma.		Watts	J	D	C	P	Volts		Ma.		Watts	J	D	C	P
	Peak	Op.	Peak	Op.	Op.					Peak	Op.	Peak	Op.	Op.				
1	0	0	50	50	?0.05	0.5	without effect			0	0	0	0		2	2	0	0
2	0	0	50	50		0.5	0.5	0	0	0	0	0	0		2	2.5	0	0.2
3	0	0	75	50	?0.05	1.0	0.5	0	0.1	50	0	0	00		2	3	0	1
4	25	25	300	100	2.5	1.5	1.5	0	...	100	100	50	50	5	2	4	0.5	1.5
5	100	100	450	100	10	1.5	1.5	0.5	...	150	140	100	100	14	2.5	5	0.5	1.5
6	200	200	550	200	40	1.5	3.0	1.0	...	250	250	200	200	50	3.5	6	1.0	1.5
7	250	250	650	300	75	1.5	5.	2	...	300	275	275	275	75.6	4	7	2.5	1.5

Coagulation:

	Low voltage				High voltage			
	T	D	Coag.	Remarks	T	D	Coag.	Remarks
1	..	0	0			0	0	
2	30″	1	1		30″	4–5	3	
3	30	3–4	2.3	Clings	12″	4–5	3	Clings
4	14	4	3	Clings	5	4	3	Clings
5	9	4	3	Clings	2	4	3	Clings
6	5	4	3	Clings	1.5	5	3	Crater
7	4	5	3	Black crater	1	5–6	3	Crater

* Investigated with needle electrode of 0.026 inch diameter; Tables I, II, III, and V with 0.014 inch electrode.
† In making deeper cuts, voltage usually falls slightly and amperage rises, depending on the depth of cut.

strain occurring across a single condenser. These lead to a coarse self-induction resonator coil, with both high and low voltage taps. The lead lines to the patient are equipped with a filter device similar to a grid leak, whereby adventitious low-frequency surges and harmonics are baffled by a proper sized condenser and their charges allowed to dissipate harmlessly slowly through the high resistance bridge.

Results: Table IV gives the results.

Comment: Cutting unit: Surgically satisfactory incisions were accomplished with 30–75 watts. Increasing the amperage in general enhanced lateral coagulation. In general increased "dehydration" meant increased wattage, largely through increased utilization of amperage. Best routine cutting was found at "high voltage, light dehydration, variometer 40." When delivered voltage rose above 225, undesirable flame burns appeared. Higher ranges of variometer dial readings could accomplish substantial fulguration, not distinguishable in surgical effect from intemperately vigorous Oudin phenomena.

Coagulating unit: In coagulating, radial penetration increased with wattage to a maximum of 3–4 mm., for the spark gap set in ordinary utilizable width; as wattage increased, the time necessary to produce maximal coagulation symmetrically diminished. In fulguration, a maximal spark of 4 mm. was obtained, but most satisfactory operative results were obtained with a 2 mm. spark at 100 v. (2 mm. penetration, with minimal carbonized eschar).

E. Commercial Spark Gap Machine (Fig. 6). This recently devised truly portable unit utilizes the same spark gap for cutting and for coagulation, the variation in condenser size being such that satisfactory coagulation can usually be obtained without resetting, merely by throwing the tumbler switch from knife to coagulation; the "coagulating" current also cuts, but with greater lateral coagulation. A reactance affords main current control, though a rather weak variometer allows some final graduation as well.

Construction: Sixty-cycle alternating current at 110 v., after passing through a 10-point reactance is stepped up by a high tension transformer and used, when cutting, to charge a single condenser creating damped oscillations by discharging through three micrometer spark gaps; when coagulation is used, however, two additional condensers are entered in series-parallel, lowering voltage and increasing amperage. A Oudin coil is activated when desired by a separate switch. A protective condenser is interposed between the variometer and the patient.

Results: The results are shown in Table V.

Comment: Again, with a gap machine, cutting appeared at about 30 watts, and was satisfactory to 75 watts; above 225 volts, flame burns appeared at the initial and parting contacts. The time varied inversely with the wattage, but lateral coagulation was not appreciably affected by amperage increase in the middle ranges.

With the switch thrown to "coagulation," cutting appeared at between 20 and 48 watts, with somewhat greater lateral coagulation than "knife," but otherwise the statements given above hold for this modality as well. In fulguration, the spark was at a fraction of a millimeter, with 0.5—1.0 mm. radial coagulation. In coagulation a 1.5 mm. radial effect was produced in 2 seconds at maximum.

In Oudin desiccation, the spark jumped maximally 4.5 mm. but could be drawn out to 6 mm. before quenched.

Low-Frequency Cutting

Since ordinary low-frequency electric currents produce burns, there is no reason why they should not also be made to counterfeit electrosurgical incisions if applied through surgical electrodes. Such currents kill, as elucidated in the introduction, but they cut identically, clinically and histologically, as do high-frequency currents.

Alternating current results are given in Table VI.

TABLE V

Major frequency when cutting (80 per cent) is 545,000, though a fainter broad band from 460,000 to 545,000 is present, and feeble oscillations appear both at 300,000 and 1,500,000. When coagulating, major frequency (about 65 per cent) is 460,000–480,000, with some at 420,000–550,000, and with the same feeble oscillations as on cutting. Oudin frequency is sharp at 706,000 per sec.

Knife:

	Voltage		Ma.		Watts Op.	Incision 3 Mm. Deep and 10 Cm. Long		Remarks
	Peak	Op.	Peak	Op.		Time	Lat. Coag. μ	
Reac. 1*	0	0	10	10	?	0		
2	100	100+	25	40	4.	Streaks, but no depth		
3	100	150	55	65	9.7	Streaks, but no depth		
4	100	150	70	85	12.8	Streaks, but no depth		
5	300	200+	110	180	36	3.5	100–300	Dry tannish brown seal
6	325	200+	130	220	44	2.2	100–300	Dry tannish brown seal
7	350	225	140	300	75	2.0	100–300	Dry tannish brown seal
8	350	225	155	300	75	1.5	100–300	Dry tannish brown seal
9			(Blows 10 amp. fuse)			1.5	200–350	Dry tannish brown seal
10			(Blows 10 amp. fuse)			1.0	200–400	

Coagulation:

	Voltage		Ma.		Watts Op.	Time	Incision Lat. Coag. μ	Remarks	Coagulation Effects			
	Peak	Op.	Peak	Op.					T.	D.	Coag.	Remarks
Reac. 1*	0	0	0	0		0	0		Spt.	desic.		
2	50	±	±	55	0.05	0	0	Sticks	...	0.5		
3	150	±	35	70	0.07	0	0	Sticks	...	0.8		
4	200	150	60	135	20.2	0	0	Streak; no cut	...	1.0		
5	325	225	75	215	48.4	3.5	200–400	Cut	3″	1.5	1.5	Cling
6	400	250	100	275	68.7	2.0	200–350	Vigorous cut				
7	425	250±	110	325	81.2	2.0	150–300	Vigorous cut				
8	450	250±	110	400	100.	1.5	200–300	Vigorous cut				
9	...		...	...		1.0	200–300					
10	...		...	...		0.7	200–400		2″	2.5	2.5	Nocling;char

Oudin:

	Volts		Ma.		Spark		Remarks
	Peak	Op.	Peak	Op.	Initial	Quench	
1	0	25	0	0	0	3	Puckers only
2	200	0	0	40	0	4	Puckers only
3	400	100	65	65	½	4½	Puckers only
4	550	400	100	100	3.5	3.5	Crater and 3 mm. zone
5	775	450	140	120	3.5	3.5	
6	825	475	160	155	4.5	5.0	1 mm. deep, 4.5 diam.
7	925	600	170	160	3.0	6.0	Carbonizes
8	975	750	180	175	4.0	5.0	Carbonizes, with a flame burn

* Variometer constantly at maximum.

Table VI

60 *cycles:* (electrode 0.023″ diameter)

Volts	Amp.	Watts	Remarks
110			Slight sputtering, no cutting.
140	0.075	10.5	Very poor cutting; 7 sec. to make 10 cm. cut 3 mm. deep.
140	0.23	32.2	Fair cutting; 3 sec. for a cut 10 cm. long and 3 mm. deep, and 11 sec. for cut 10 cm. long and 1 cm. deep.
220	0.5	110	Good cutting to any depth, 10 cm. long in 3 sec.; only 100–300μ lateral coagulation.
500	0.5	250	Excellent quick cutting; no flame; only 100–300μ lateral coagulation.

In view of the foregoing, *direct current* was tried, using the large Cruft Laboratory battery in order to eliminate any possibility even of commutator ripple; no other leads or taps were in circuit. The cutting electrode was negative and cool. The results are shown in Table VII.

Table VII

Volts	Amp.	Watts	Remarks
120			No cutting; very slight coagulation about the needle point.
180	0.30	54	Good cutting, 4 sec. for 10 cm., cut 3 mm. deep.
240	0.24	57.6	Excellent cutting to any depth, 3 sec. 10 cm. long and 3 mm. deep; 4.2 sec. (144 watts) for 10 cm. cut 1 cm. deep; only 200–500μ coagulation lat.

With polarity reversed the needle became red to white hot during cutting, and sparking and searing were more marked; amperage readings were also 25 per cent higher.

Some photomicrographs of these cuts are given in Figures 7 and 8 for comparison with ones of analogous wattage in high-frequency currents. As larger electrodes are used the zone of carbonization widens, hemosiderin appears in the capillaries in the proximal zone of coagulation, steam vacuoles are larger and more frequent, and tissue is more disrupted physiomechanically. Carbonization is not apparent microscopically in incisions with the negative pole electrode.

Comment: Here, with a slightly larger electrode than in experiments detailed before, incisional effects could be produced at 10 watts, but surgically satisfactory cutting was not obtained until about 30 watts were delivered; furthermore, sluggish but adequate incisions could be affected at 140 v.; at 220 v. however effects were consistent and free. It is notable that flame burns did not appear at low frequency even at 550 v.; nor with direct negative current up to 240 v. (though with polarity reversed flaming and sparking spoiled every incision).

Histologic Findings

General: Cuts made by any of these electrosurgical machines have shown common general characteristics histologically; different modalities and different machines produce varying depth of zones, or occasionally omit a zone, but a recent electrosurgical incision is readily recognizable as such. These general divisions, illustrated in parenchymatous tissue, commencing at the face of the incision and thence progressing in penetration are:

Carbonization: 1–15μ. Black and brown common granular resolution of all structure; easily wiped away even by the passage of the electrode; frequently wholly absent in surgically excellent incisions. Usually significant of excessive voltage.

*Hyalinization:** 6–50μ. A formless, slightly granular, deep pink-staining, molecular disintegration in which rare distorted nuclear or cystoplasmic outlines may be suggested, but predominantly utterly without structure.

Desiccation: 5–30μ. Corresponds to the mummification necrosis of Clark, Morgan and Asnis[8]; essentially an extraction of fluid from cells. Cytoplasmic and nuclear

* Though this is accurately descriptive, the change indicated by this term has only apparent resemblance to the chronic change called hyaline degeneration.

outlines are intact, but considerably collapsed in varying degree upon themselves; vivid staining; not mere pyknotic change. Throughout this, *and the zone of coagulation*, steam vacuoles, sometimes containing a small amount of precipitated albuminous fluid, are found in varying abundance and size, if the structure of the tissue is such as to allow their formation.

Coagulation: 20–700μ. Classic coagulation necrosis, with intact or rarely pyknotic nuclei and vitreous deeply-staining cytoplasm. Blood cells within capillaries contained in the distal half or two-thirds of the zone are intact and unharmed. Rarely, if electrode movement has been sluggish, the coagulum in the depths of the incision will have shrunk somewhat, freeing occasional individual parenchymal cells into microscopic lakes of plasma intervening between this and the next layer; primary healing is then dubious.

Viable: 5–250μ. A zone containing occasional rounded cells, and a few with deeply-staining cytoplasm; nuclei and chromatin network everywhere however are essentially unaltered, and this zone is viable.

TABLE VIII

			Carbon	Hyalin	Desic.	Coag.
A.	270,000	1	9–11	6–10	6–10	180–200
		2	6–10	10–12	?o	150
		3	3–5	3–8	?o	100
	1,060,000	1	30	20–30	15–45	240
		2	15	25–30	10–60	180–300
		3	3–6	20–25	30–60	120–150
		4	10–15	15	30–45	60–90
		5	0–10	?5	30	50–60
	4,600,000	1	6–8	16–20	75–100	150–200
		2	3–6	15–18	30–50	125–150
		3	2–4	15–18	30–45	40–150
		4	2–4	12–15	15–45	60–240
B.	Fast	4 Small	0–2	18	10–18	6–12
		Large	0–2	18	6–18	30–90
	Slow	4 Small	1–3	3–4	8–10	6–100
		Large	4–8	6–12	4–10	90–120
C.	Voltage control	2 6 knife	6–9	3–6	3–6	50–100
		3	3–6	3–6	0–3	3–40
		0	0–3	0–3	0–3	30–100
		3	0–15	0–15	20–40	120–150
		6 coag.	?	+	25–30	180–200
	Voltage control	4 6 knife	6	3–6	?–3	70–80
		3	6–9	3–6	0–3	120–150
		0	6–9	10–40	30–40	170–190
		3	6–9	15–20		250
		6 coag.	10–15	12–15	30–40	200–225
D.	Low voltage	50 Heavy	15–18	6–30	0–4	150–250
		Med.	3–6	20–30	3–15	160–200
		Low	0–3	15–20	15–30	130–160
	High voltage	50 Heavy	15–20	10–20	3–6	300–450
		Med.	10–14	10–15	?	300–350
		Low	20–30	20–30	?	200–350
E.	Variom., Max.	10 knife	9–12	20–30	40–60	150
		7	6–9	15–30	10–30	110–120
		5	0–3	?	?	80–90

Normal: Beyond this.

Performance: For purposes of comparison the microscopically determined zones of histologic alteration, in microns, produced by various machines and different dial settings, are tabulated as shown in Table VIII.

Previous personal experimentation, and confirmatory investigation here, have shown that uncomplicated primary healing in well-vascularized parenchymatous tissue may be expected to occur if the total zone of lateral damage on one side of the incision does not exceed 250–300 microns; above this primary healing is dubious; and above 600–700 microns secondary intention or even occasional sterile abscess (which may be wholly absorbed) is almost inevitable. It must be realized that these statements are generalities, and that other factors enter into healing than the initial (and major) incisional damage.

The incisions tabulated in Table VIII and illustrated in Figures 9–12 were 10 cm. long and 3–4 mm. deep, and were made with the electrode moving at maximal speed for the dial settings given, without sluggishness or hesitation.

ELECTRODES

If cutting effects are a function of current density, electrode size should exert appreciable effect. The customary needle point used in electrosurgery has a mean hilt diameter of 0.025–0.028 inch; this point is sturdy and mechanically sound. A steel wire of 0.013–0.015 inch diameter is more flexible; for satisfactory use therefore it cannot be more than 1.5–2.0 cm. long, but this length active tip can be welded onto a mechanically sturdier base. It was thought that the density effect might be reflected in wattage, but it was found rather to be reflected in time. Example (Table IX).

Frequency 1,060,000		Volts		Ma.		Watts	Time
		Peak	Op.	Peak	Op.	Op.	
Tap 2	0.013 inch	650	225	260	110	24.7	4.0
	0.026 inch	650	225	260	110	24.7	5.5
Tap 3	0.013 inch	425	180–200	170	85	15.1	6.0
	1.026 inch	425	175–50	170	85	14.9	9.0

It is claimed by a manufacturer that the use of an alloy of tantalum and tungsten as an electrode avoids completely the cling of carbonized tissue to the needle; this was not investigated.

HEMOSTASIS

An attempt to present experimental protocols here is unwarranted, because of the diversity of modifying factors. The chief utility of endothermy, however, lies in its control of capillary bleeding and in lymphatic sealing. Frequency was found to exert no appreciable effect on this, and, within the range investigated (270,000 to 4,600,000), any wattage that produced incision was also initially hemostatic. However, there was variation in plasma-ooze through the walls, late beady formations of blood in a gaping incision, primary control of venules and even arterioles, and degree of manipulation which an incision would stand without loss of hemostasis. The degree of effective stasis in general seemed more nearly to parallel the combined width of the zone of hyalinization and desiccation than to show a consistent relation to the width of the zone of histologic coagulation. Hemostasis of larger vessels effected by electrocoagulation (contact) or by fulguration was histologically merely an intensification of essential incisional processes, with punctate risk of chances of primary healing, due to carbonization, eschar or relatively extensive coagulation necrosis. Since the breadth of zones of hyalinization, desiccation and coagulation is mainly dependent upon amperage, it follows that devices with a greater reservoir of deliverable amperage (within adequate voltage limitations) have roughly, other factors being equal, greater surgical resource.

ARCS

In Figures 13 and 14 are reproduced a few instantaneous photographs (1/200 sec.) of various discharges produced by some of the machines investigated. They are in-

dicative of the probable reasons for the different actions of various types of endothermic current. Figure 13 depicts non-cutting endothermic currents; their broad, brush- and tent-like discharges disperse heating effects irregularly over relatively large areas (*a, c, d*) or else wave frond-like with great rapidity from one microscopic area to another (*b*). Those of cutting currents, however (Fig. 14), are seen to be, for the most part, compact, stubby, slim pillars; the energy released is concentrated sharply in one area; the density of the current is high. The cutting effect is not the result of a new property of wave motion inherent in undamped oscillations; it is not even dependent on oscillations (pp. 435, 437), though high frequency oscillations are necessary for its production in the *living* subject.* At the time cutting effects appear in contact application, a new factor absorbing energy enters (as, e.g. Table II, *d*) the maintenance of an arc, that is, the persistent ionization of the track of current between electrode tip and the tissue.[15] It is the persistent ionization of this tract that prevents dispersion of the current as brush discharge and causes the intense concentration of heating effect (electric arc) whose results under proper conditions is electrosurgical incision. Figure 14 *c, d,* shows in exaggeration the character of the discharge which produces cutting; Figure 14 *a, b,* is an illustration with excessive wattage; and Figure 14 *e,* shows ordinary conditions with an infinitesimal arc which can be seen from above, in the dark, as the electrode advances.

SUMMARY AND DISCUSSION

Adequate current density is the sole general requirement for cutting by electricity. This involves maintenance of an electric arc between the active electrode and the tissue. *Direct current* and *low-frequency current* can produce true incisional effects as well as can high-frequency oscillations of either damped or undamped form. Using surgical electrodes, with direct or low-frequency current, approximately 150 v. are required, and 35 to 150 watts, for an incision of ordinary surgical depth (3 to 10 mm.); the amperage drawn is roughly proportional to the depth of the incision. The function of high frequency in electrosurgery is to afford the non-lethal passage of the necessary current through the living body; its direct participation in incisional effects is nil. In pioneer work it was easier technically to maintain adequate current density with undamped than damped current, but mysterious effects are resident in neither. With *high-frequency current* approximately 225 v. are required for incisional effects, and 18–25 (undamped oscillations) or 30–75 (damped oscillations) watts; the comparative time required for incisions was a rough function of wattage, and the degree of lateral coagulation tended to parallel the delivered amperage (about 1–2 ma. per micron). The critical voltage for cutting effects with high frequency is about 220, and at about 250 undesirable flame burns at the lips of the incision begin to appear.

Electrosurgical coagulation is effected by contact application at voltages less than 200, the depth of coagulation again being a function of delivered amperage; if voltage is too high, desiccation and carbonization occur about the electrode, with cessation of deep penetration. Electrocoagulation can also, however, be effected by otherwise lessening the density of the cutting current (substitution of brush-discharge for true electric arc) through the use of electrodes of greater presenting area (hemostat, forceps, ball, etc.).

An ideal machine should furnish the foregoing amplitudes flexibly *at the electrode tip;* it should not include expensive, unnecessary and harmful voltages on its routine dials; it should have 250–300 ma. delivered capacity, most of which should be electively utilizable at below 200 volts. Its current should be smoothed free of harmonic faradic effects by a proper grid-leak type filter; its frequency should be low enough

* This takes distinct issue with (15), p. 1866, par. 4 and 5.

that (1) conduction delivery by clinically adequate cables is possible and (2) uninvolved metal clamps and retractors in the operative field do not warm; all parts of its circuits should preferably be grounded through supply wiring. A keyed Oudin coil for high voltage requirements might be available. The control panel should be horizontal and simple, and should have sturdy detachable boilable switch and dial-handles raised a decimeter above the panel surface. Many of the triode machines on the market today find difficulty in supplying adequate amperage without excessive voltage; and many gap machines, supplying adequate amperage, also include unused dial possibilities of redundant voltage. Finally, present cartel prices of most machines, in view of their actual equipment, wiring and labor, are excessive; and those of several of the pioneer machines remain prohibitive.

CONCLUSION

Electrosurgical incision is shown to be independent of frequency or wave form, and to depend upon current density. The characteristics and performance of an experimental unit, and several commercial machines, are examined; requirements of an ideal machine are deduced.

It is a pleasure to acknowledge the suggestion and guidance of Mr. H. R. Mimno, of the Cruft High Tension Laboratory, Harvard, in construction of the experimental unit; I am indebted to Professor G. W. Pierce for use of laboratory facilities there, and to Messrs. Carley and deYoung for their assistance. Dr. Richard Light kindly extended use of the Laboratory of Surgical Research, Harvard, and Misses G. E. Howard and M. L. Phillips assisted in the histology. The investigation was conducted under a grant from the DeLamar Mobile Research Fund, Harvard Medical School. The Children's Hospital, Peter Bent Brigham Hospital, and Mr. Charles H. Kehlenbach, were kind enough to allow temporary use of some of their electrosurgical devices. Wiring diagrams given depict the conditions in the machines investigated, and have been made independently of manufacturers literature in the rare instance where such information is published. Dr. George A. Wyeth, Dr. Grant E. Ward, and Mr. K. Stoye have been helpful with information and criticism.

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[For Remainder of References see p. 459.]

SKELETAL TRACTION & PRESSURE IN TREATMENT OF FRACTURES*

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INTRODUCTION

THE recognition of fractures and their treatment are subjects which have engaged the attention of medical practitioners in all ages, and the evidence of Egyptologists proves that many thousands of years ago broken bones were tied to splints in much the same way as they are today (Hey Groves).

With the increasing number of fractures, a toll for the more rapid means of transportation enjoyed in this age, we find altered methods of treatment offered in an effort to obtain a better percentage of good anatomical and functional end results than has been permitted by other means at our command. Skeletal traction has become a most popular method and the applicability, the efficiency, and the simplicity connected with its use sufficiently recommend it to the careful surgeon.

The object of this thesis is to review the treatment of fractures of long bones of the extremities as it has been carried out by various means of applying skeletal traction and pressure, on the Orthopedic Service at Harborview Hospital.

The combination of the methods of Codivilla, Steinmann and Becker is most popularly known now as the Steinmann pin method of skeletal traction. Our description and case reports has been limited to fractures in which the Steinmann pin was used for traction and also certain other cases for which specially devised apparatus was employed to exert pressure in lateral displacement of the fragments.

We have divided this paper into several sections:

(1) A review of the anatomy of the joints, with special reference to the application of metal traction and pressure apparatus

(2) Descriptions and photographs of methods and means by which skeletal traction and pressure have been applied

(3) Case reports, with photographs

(4) Summary.

ANATOMY OF THE JOINTS WITH REFERENCE TO THE APPLICATION OF SKELETAL TRACTION

Skeletal traction may be applied at the following sites:

1. To the os calcis
2. To the lower end of the tibia
3. To the upper end of the shaft of the tibia
4. To the lower end of the femur above the condyles
5. Through the olecranon process of the ulna.

There are less common sites for pin application but these locations will suffice for all fractures of the extremities except those of the forearm. Transfixion is not advisable at the lower end of the forearm bones because of the liability to inflammatory adhesions of the tendons around the wrist joint. Skeletal traction may be used in certain cases of fractures of the phalanges and then a small wire is preferable (Scudder).

OS CALCIS. The *os calcis* (Fig. 1) was one of the first bones to which skeletal traction was applied and this is quite efficient for the great majority of fractures below the knee. The pin is best applied under local anesthesia, at a point three

* From the Harborview Hospital, Seattle, Washington. Thesis submitted to the Faculty of Surgery of the Graduate School of Medicine of the University of Pennsylvania in partial fulfilment of the requirements for the degree of Master of Medical Science (M.Sc.(Med.)) for graduate work in Surgery.

fingers breadth below and behind the external malleolus, from without, inwards and at right angles to the bone. Owing to the late development of its bony structure, the os calcis is the only bone which cannot be satisfactorily employed for skeletal traction in children under twelve to fourteen years of age. The danger of injuring vessels, nerves or tendons in this location is negligible.

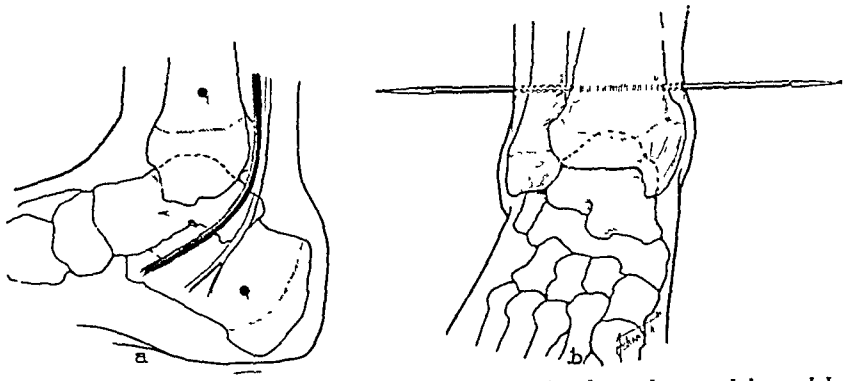


Fig. 1. A. Lateral view of ankle joint showing sites for placing pin through os calcis and lower end of tibia. B. Anteroposterior view of ankle joint showing pin through lower end of tibia.

Lower End of the Tibia. This site (Fig. 1) has become more popular of recent years for fractures which occur in the pelvic bones, femur, tibia or fibula in which skeletal traction is to be employed. The pin is applied from within outwards, at a point about two fingers breadth above the tip of the internal malleolus. The pin is inserted with the point directed outwards at right angles to the shaft of the tibia and following a plane which will make the point of exit slightly anterior to the point of entrance. The fibula is posterior to the pin and is not penetrated by it. The anterior and posterior tibial vessels and nerves to the foot will be avoided, but there is danger of injury to the tendons. The articulation of the astragulus with the tibia which causes a concavity in the lower end of the tibia is also avoided. The epiphysis which does not undergo complete fusion until the nineteenth year is safely avoided if this area is used in children.

Upper End of the Shaft of the Tibia. The pin is inserted just below the tibial tubercle and about two fingers breadth lateral to it (Fig. 2), thereby missing the epiphysis which does not unite with the shaft until about the twenty-second year of life. The fibula is posterior and is not penetrated at this site.

Lower End of the Femur. Skeletal traction is applied in this location by selecting a point on the mesial surface of the thigh just above the tip of the internal condyle or adductor tubercle (Fig. 2). Consideration must be given here for the lower epiphysis which unites with the shaft of the femur from about the twentieth to twenty-third year of life. The synovial membrane of the knee joint extends onto the femur, embracing approximately one third of the circumference of the shaft anteriorly, and must be carefully avoided to prevent joint complications. The popliteal vessels and nerves are in fairly safe territory. The introduction of the skeletal traction may be accomplished also, from the lateral surface just above the external condyle.

Olecranon Process of the Ulna. Occasionally fractures of the humerus may be more easily controlled by a pin through the olecranon process of the ulna (Fig. 3). The arm is flexed to a right angle and the pin or wire put through the olecranon just below the head of the radius and in front of a line drawn along the anterior border of the humerus. The joint cavity is avoided, the radial nerve is at a safe distance, and the important vessels are well anterior. The radius is not penetrated. The olecranon

process does not unite with the shaft until about the fourteenth year and should not be used before this age.

ether, and painting with iodine. The area is then anesthetized by injecting a sufficient quantity of 2 per cent novocaine at the

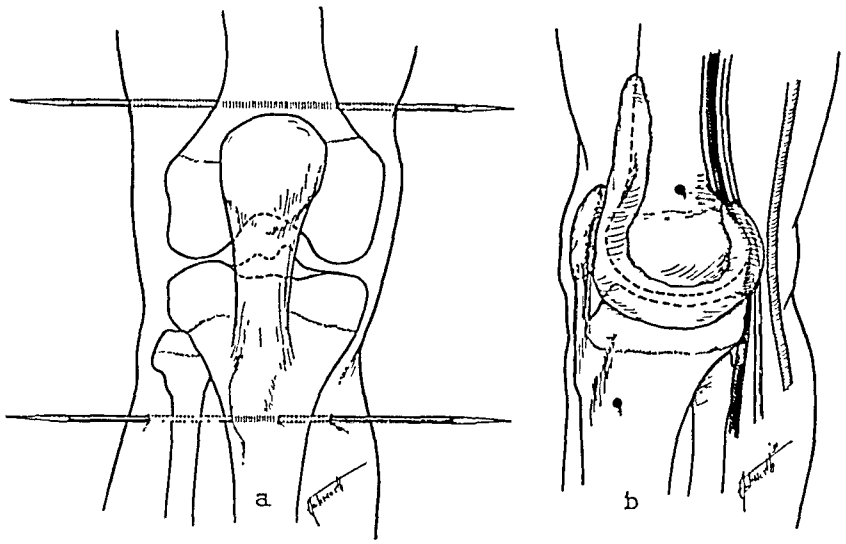


FIG. 2. A. Anteroposterior view of knee joint illustrating pin through upper end of tibia and lower end of femur. B. Lateral view of knee joint showing sites of pin application.

METHODS OF APPLYING SKELETAL TRACTION

STEINMANN PIN. The pin is a chromium plated solid rod, not divided, varying in length from 4 to 10 inches and having a diameter of $\frac{5}{32}$ of an inch (Fig. 5, A). The divided pin is constructed to permit the two ends to be separated by unscrewing it in the center and withdrawing each end. In this way there is claimed to be less chance for infection than when the solid pin is pulled back through the bone. These divided pins are weak and break in the middle portion. We have had no trouble with infection by using the solid pin and we have had the divided pin break under pressure so that we no longer use it. One end of the pin is sharp and the other dull, with bevelled sides to fit in a small handle (Fig. 4, B), with which it is pushed through the bone by a boring motion. The pin is prepared for use along with the other instruments, the ends of the pin being protected by placing corks over them.

The skin is prepared by shaving, washing with green soap and water, scrubbing with

point of entrance and exit of the pin. The site of fracture may also be injected with 20 to 40 c.c. of the same strength novocaine according to the method popularized by Böhler. Strict surgical asepsis must be maintained during this procedure and the technique must be faultless to prevent infection. The excess of iodine is carefully removed with alcohol to prevent skin burn, and dressings are applied over each end of the pin. Two gauze pads about 3 inches in diameter are used and held in place by twisting another larger pad over the pin ends. Sheet wadding is wrapped around the limb to keep the dressing close against the skin. It is not advisable to use gauze bandages or adhesive for this purpose because later swelling of the extremity may occur and the increased tightness of the tape or bandage may cause circulatory disturbances.

TIME AND MANNER OF REMOVING THE PIN. The time for removing the pin depends upon the age of the patient, the location of the fracture, callus formation and the occurrence of infection. Immobilization with skeletal traction is usually

continued a shorter time in children and a longer time in very old patients. If infection occurs, the pin is immediately removed and the infection treated locally. If callus formation is delayed, the pin is left in place for a longer time. In general the following schedule may be observed: fractures of the neck of the femur, twelve to fourteen weeks; fractures of the pelvis, four to six weeks; intertrochanteric fractures, seven to nine weeks; subtrochanteric fractures, six to ten weeks; fractures of the shaft of the femur, five to ten weeks; fractures of the tibia, four to twelve weeks; operative cases, four to fourteen weeks.

The patient is not aware of the presence of the pin unless told. Pin wounds never require redressing prior to removal of the pin. Practically all of our pins are embedded in plaster casts for rigid immobilization. If the cast is not removed at the same time decided upon for removal of the pin, a hole 3 to 5 inches in diameter is cut on each side of the cast around the pin. All sheet wadding and dressings are removed. Loose plaster is swabbed from the pin with the aid of sterile water and gauze pads. The protruding ends of the pin and skin areas surrounding them are cleansed with ether and iodine and the pin is pulled out with a pair of pliers. Sterile dressings are applied to each side of the extremity and they need not be changed for several days. Healing takes place within two weeks.

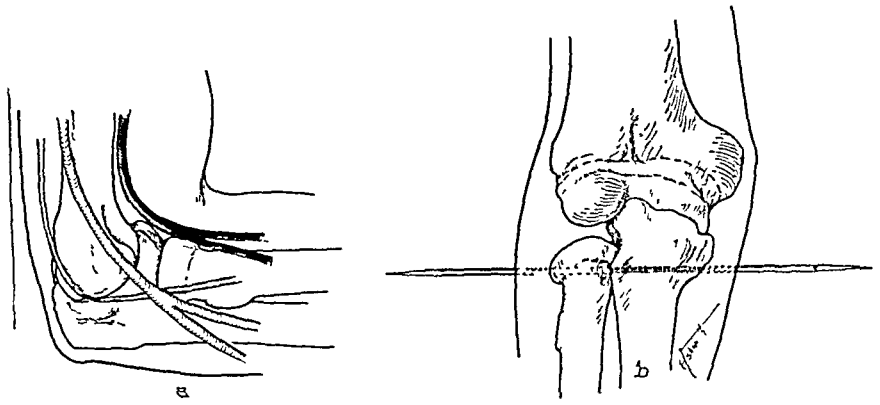


Fig. 3. A. Lateral view of elbow joint showing site for inserting pin through olecranon process of ulna. B. Antero-posterior sketch with pin in place.

"U" Stirrup. This device (Fig. 4, A) permits traction to be exerted on a Hawley table or by any other means available.

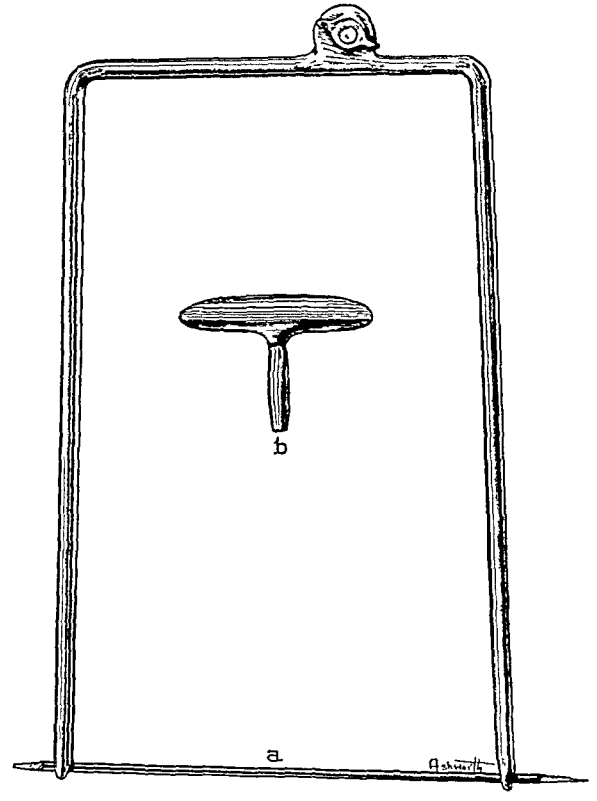


Fig. 4. A. "U" stirrup for traction, constructed so as to permit tips of pin to be slipped into either end of "U." B. Handle for manual insertion of pin.

After extension has been accomplished, the pin may be incorporated in a plaster cast which then maintains traction on removal of the stirrup.

Steinmann Pin Handle. This handle (Fig. 4, B) is used for manual insertion of

the pin. The bevelled tip of the pin is held firmly in the hollowed end of the handle while a boring motion is used for inserting shortening, and in certain spiral fractures where reduction is difficult to maintain, the sliding sleeve type of pin may be

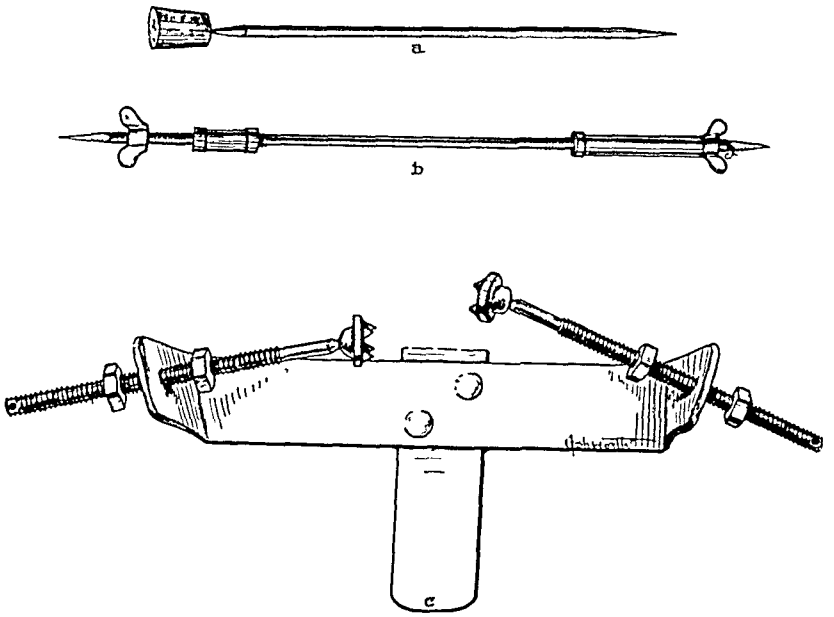


Fig. 5. A. Steinmann pin, sharp end protected by cork. B. Modification of Steinmann pin with sliding sleeves and wing nuts for exerting and maintaining lateral pressure. C. Lateral pressure clamp.



Fig. 6. Adjustable metal splints for use with double pin skeletal traction.

the pin.

Steinmann Pin with Sliding Sleeves. A modification of the Steinmann pin is shown in Figure 5, B, with threads on either end extending about one-third the length of the shaft. After the pin has been inserted in the usual manner, the sleeves are placed over the pin ends and pushed against the bone through small incisions in the skin. Pressure is applied and maintained by wing nuts screwed onto the pin. The pin was devised by Roger Anderson and used successfully in fractures where lateral pressure and traction are both necessary. Case IV represents the type of cases where this apparatus is of value. In general, where there is a lateral displacement as well as applied and buried in the cast for rigid immobilization.

Lateral Pressure Clamps. This apparatus (Fig. 5, C), also devised by Dr. Roger Anderson, was used to exert lateral pressure in a patient with a fracture of the head of the tibia. The pronged pressure points on a universal joint, attached to the adjustable rods, were placed against the bone fragments on either side through small incisions made in the skin. Pressure was exerted by placing a pin in the holes at the end of the rods and screwing them down. The pressure was fixed by nuts screwed onto the rods and placed on either side of the frame. The small incisions were closed with dermal suture and the entire appara-

tus was then incorporated by a plaster cast extending from beyond the toes to the upper thigh.

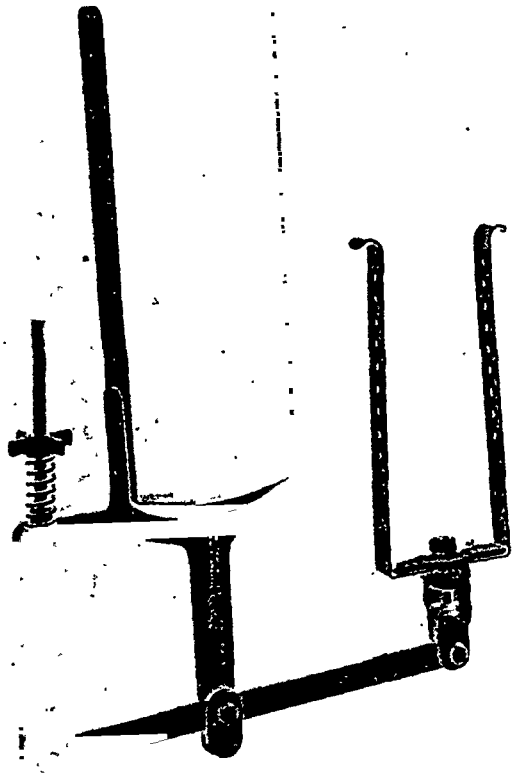


Fig. 7. Skeletal and well-leg countertraction splint (Roger Anderson).

Extension Bars for Double Pin Skeletal Traction. An apparatus was described by Abbott to aid in the fixation of bones in the leg following operative lengthening of the tibia and fibula. His suggestion has been utilized in the two bars, Figure 6, which permit the use of two pins for skeletal distraction. Each bar consists of a hollow metal tube 8 inches in length with a hole drilled transversely through the tube, ½ inch from the closed end. In the other end of the hollow tube was fitted a threaded rod, smaller in diameter but equally long, with a hole drilled transversely through its free end and with a nut for adjusting and fixing the length. These bars were used with two pins, one through the upper and one through the lower end of the tibia, and the bars were attached to the pins on each side. Distraction force was exerted by turning the nut on each bar and the fracture reduced. The pins but not the

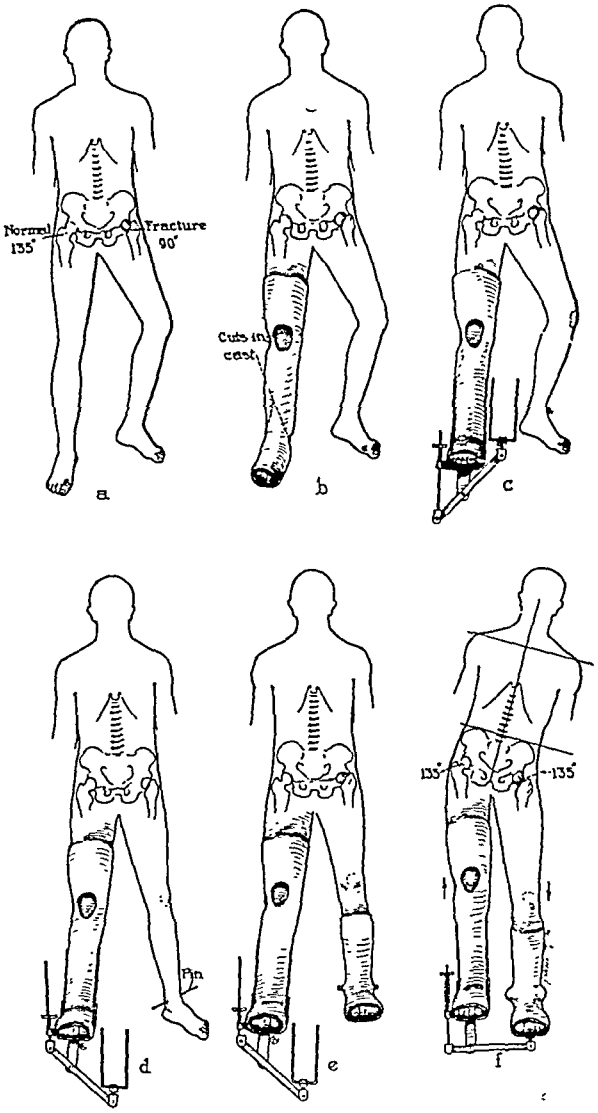


Fig. 8. Steps in routine application of well-leg countertraction splint. A. Before reduction in fracture of neck of left femur. B. Application of cast to mid thigh of well leg. C. Incorporation of splint in cast. D. Pin inserted through, tibia of injured leg. E. Pin incorporated in cast extending to knee. F. Splint attached, rotation adjusted, traction applied. Note angles at hips after reduction, with normal relationship maintained between pelvis and spine.

bars were incorporated in a plaster cast extending from the toes to the middle of the thigh. Should the fracture require manipulation later, a circular cut is made all around or half way around the cast in the plane of the fracture. Lengthening the distraction bar by turning the nuts on one

or both sides will overcome lateral displacement of the fracture. Tongue blades may be inserted in the cut in the cast if the bars are not used, and the same lateral displacement may be overcome. This procedure makes a V-shaped separation in the cast and is called "wedging." The cast is repaired with a few turns of plaster bandage around the cut. The use of these bars is further described and illustrated by Case V and Figure 12, B.



FIG. 9. Case II. A. Before reduction B. After reduction with well-leg countertraction splint. C. One month later. D. Two months later.

SKELETAL AND WELL-LEG COUNTERTRACTION SPLINT. This appliance (Fig. 7) was devised by Dr. Roger Anderson of Seattle. It is made of aluminum and weighs approximately 2 pounds. The essential parts are attached to a frame or base on one side of which is a countertraction stirrup, this being incorporated into the cast of the well leg. A transverse lever arm is pivoted on this stirrup, the axis of which is several inches distal to the sole of the well foot. On one side of the lever is connected a threaded rod, on which a nut, turned by the fingers alone, furnishes the traction force. A coil spring under the traction nut is for the purpose of maintaining a smooth, flexible and yet ever constant traction.

The opposite end of this lever arm, which is underneath the foot of the injured leg, is attached to a U-shaped band, the traction stirrup, with perforated holes ½ inch apart. The tongs, pin or wire are connected to this lever by special devices. The attachment for the use of pins is shown in Figure 7. The rotation of the fractured extremity is accomplished by a screw lock located at the base of the traction stirrup.

Technique of Application. This method of treatment is applicable to all fractures of the femur, including intertrochanteric and subtrochanteric fractures of the neck and fractures of the shaft; to fractures and injuries of the pelvis; and to certain fractures of the tibia. Moreover, it is a great aid in providing both traction and im-

mobilization for conditions of non-union or malunion and following reconstruction operations.



A B

Fig. 10. Case III. A. Fracture of lower third of left femur before reduction. B. Same, showing pin in femur, two months after reduction.

An *x*-ray examination requiring two views or stereoscopic films generally precedes treatment. The patient need not be taken from the cart on which he entered the hospital for either roentgenograms or the application of the splint.

A hypodermic or 1/8 to 1/4 grain of morphine sulphate is usually given on admission to the hospital. Both legs are cleansed and any wounds dressed. All necessary manipulation should be done while an assistant makes gentle traction on the leg, in order to decrease pain and lessen trauma.

Nearly all of the injuries for which this method is applicable may be treated according to a standard routine. The following is an outline of the procedure. (Fig. 8, A–F.)

(1) Apply cast to mid thigh on well leg

(2) Incorporate splint in cast

(3) Insert pin through tibia

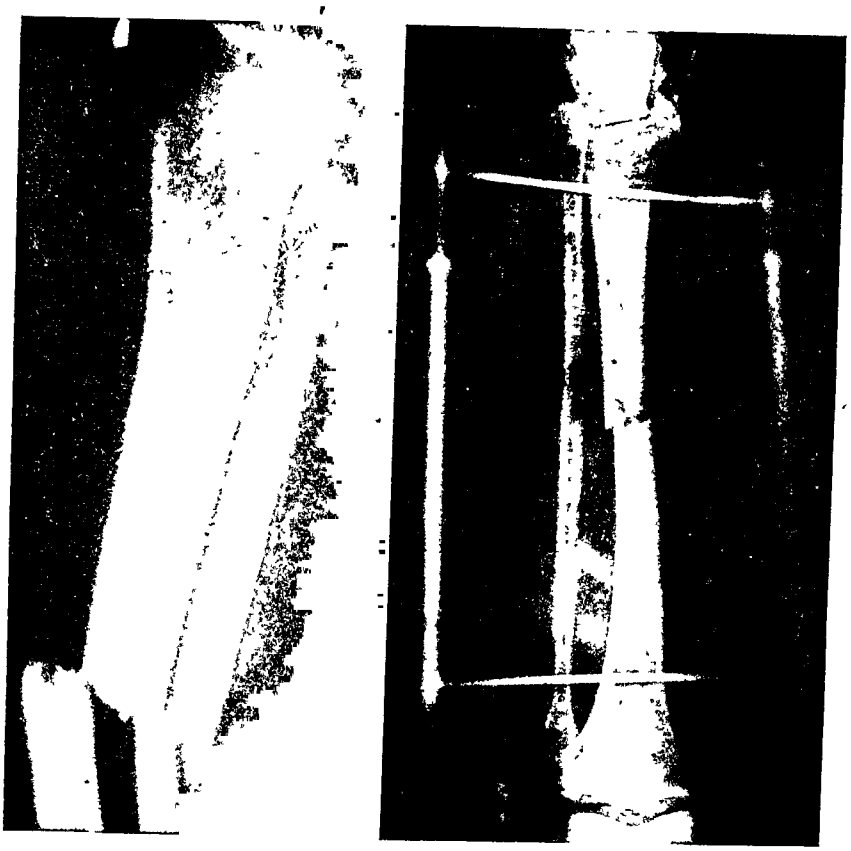
(4) Apply cast to injured leg, incorporating pin

(5) Incorporate splint in cast, exert traction, adjust rotation, complete reduction.

Application of Cast to Well Leg. The application of the cast is a very important procedure. First, stockinet is applied, then the leg is well padded with sheet wadding, especially in the region of the heel and ankle. A thin flexible piece of felt is applied to the sole of the foot. The leg is held in adduction, the knee in complete extension, the foot at right angle and the ankle in slight varus, while the plaster-of-

Paris cast is applied, extending from one inch beyond the toe-nails to mid thigh.*

Previous to the completion of the cast a equal amount of space on each side, to avoid pressure by the splint on either malleolus. A space of at least a half inch



A B

FIG. 11. Case IV. A. Comminuted fracture of lower end of left femur, before reduction. Anteroposterior view. B. Same, after reduction using Steinmann pin with lateral sleeves for pressure.

thick reinforcement of plaster is applied to the sole of the foot, made thicker under and beyond the toes and over the plantar surface of the heel. To secure a balanced distribution of counterpressure throughout the plantar surface of the foot, that portion of the cast must be accurately moulded to fit the sole. The cast is cut out over both malleoli, and over the dorsum of all the toes. By the time these holes are cut, usually the plaster is set sufficiently to permit attaching the splint to the cast by a plaster bandage.† The base of the splint (Fig. 7) is set at a right angle to the longitudinal axis of the leg at a plane through the center of the malleoli, with an

*See Anderson,[4] Figure 6A.
†See Anderson,[4] Figure 6B.

must be preserved between each malleolus and each side of the countertraction stirrup.

The technique of pin insertion has been described on page 445, and illustrated in Figure 4.*

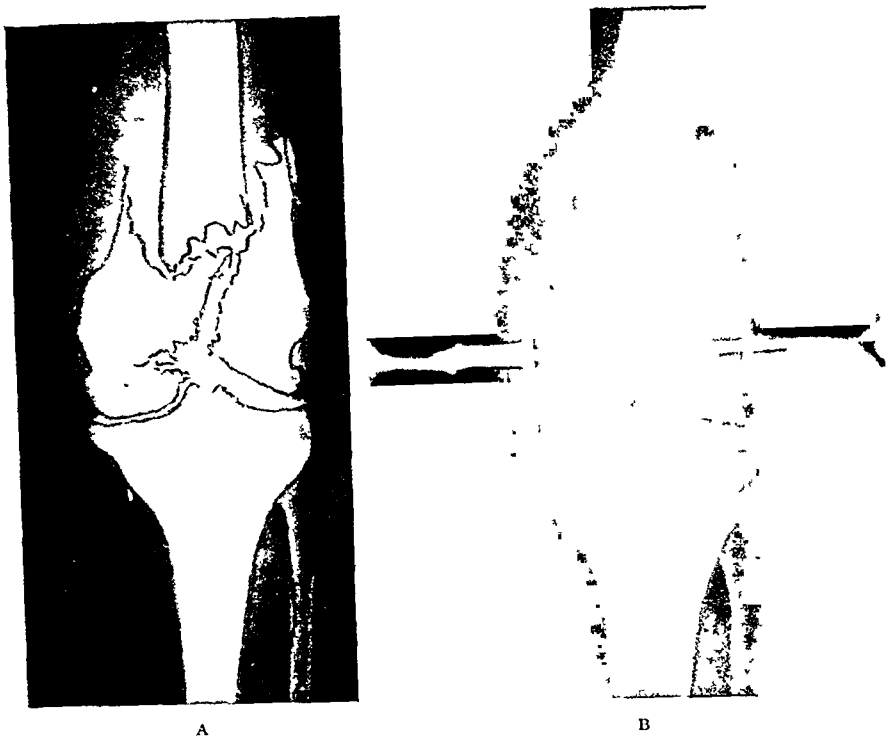
Application of Cast to Injured Leg. The leg should be well padded with sheet wadding, especially over the malleoli and heel. A cast, incorporating the pin, is applied to the injured leg extending from one inch beyond the toe-nails up to a level 5 inches below the knee joint.† The sole of the foot is reinforced with plaster. Openings are cut in the cast over both malleoli.

The ends of the pin are then put through the perforations in the traction stirrup, the

*See also Anderson,[4] Figure 6C.
†See Anderson,[4] Figure 6D.

holes being selected which will enable the splint to be set up closely against the plantar portion of the cast. The pin ends are corked,

Correct rotary alignment is obtained by rotating the fractured leg internally or externally and is held by tightening the



A B

Fig. 12. Case v. A. Comminuted fracture of middle third of tibia and fibula. B. Same, after reduction, showing double pin distraction apparatus in place.

and both corks and stirrup are incorporated in the cast, preferably by a single plaster bandage.*

The next step is to exert traction by screwing down the traction nut. When the lever arm is at a right angle to the longitudinal axis of the well leg, sufficient traction has usually been exerted.

As traction displaces the acetabulum downward on the injured side, the patient prefers to lie on the bed with the upper part of the body toward the injured side, a position he should be encouraged to assume. From a cursory glance the pelvis may appear tilted, but closer examination clearly shows no abnormal relationship between spine and pelvis and consequently no danger of list or scoliosis, because the spine sets at right angles on the pelvis (Fig. 8, F). Abduction, therefore, is obtained in a physiological manner at the hip and not by a pathological list to the pelvis.

*See Anderson,[4] Figure 6E.

rotation nut at the base of the traction stirrup which was applied to the injured leg.*

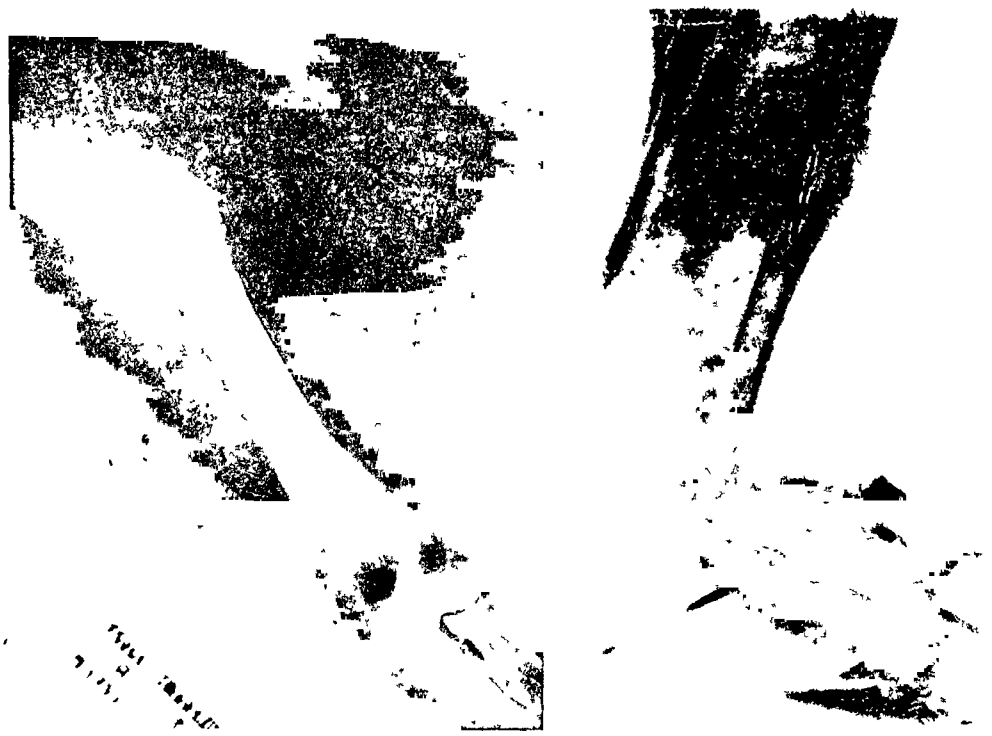
Reduction should now be checked by roentgenograms, traction and rotation being modified as indicated. If the plaster has set sufficiently the cast may be cut over the posterolateral aspect of the head of the fibula and also over the anterior surface of the knee joint. Usually this trimming of the cast is not done for several hours or until the next day and while the labor involved in cutting the cast is greater, the incidence of breaking and twisting of the cast is lessened. Occasional development of pressure points in these regions has made this a routine procedure.

The patient is not only permitted to sit up but is encouraged to do so and to use the trapeze freely; in a day or so he is able to raise himself up for toilet necessities. If the patient with a hip fracture is not in a

*See Anderson,[4] Figure 6E.

condition of shock, he will be permitted to use the wheel chair on the second or third day. Some patients, especially those with heart lesions, can safely be allowed to sleep in a semi-upright position. Patients with hip and intertrochanteric fractures may be turned on the abdomen daily. The greater part of the anterior portion of the cast of both legs may be removed, except for three transverse bands 2 inches in width.

After-care is at a minimum, for very little needs to be done except occasionally to cut out over the heel or to split the upper rim of the cast. The nurses and also the patients should be directed to massage and move the knee caps daily. It is advisable to repeat the roentgenograms as indicated. If the second plate reveals good position, the patient may be sent home. No attempt is made to dress the pin wounds until the time arrives for removal of the pins which will be from a few weeks up to a period of sixteen weeks, depending upon the location and type of injury.



A B

FIG. 13. Case VI. A. Comminuted fracture of middle third of right humerus before reduction. B. Same, after reduction, bone ends wired together and alignment maintained by pin through olecranon.

CASE REPORTS

CASE 1. E. H., female, aged eighty years. *Diagnosis:* Extensively comminuted intertrochanteric fracture of the neck of the right femur, with the lesser trochanter displaced slightly mesial.*

Patient was a senile individual from whom an adequate history was not obtainable. She had apparently fallen on the floor of her home and injured her leg.

Because she was unable to cooperate, a few drops of anesthol were administered and the well-leg countertraction splint applied with a cast on the left leg extending to the thigh. The cast was cut out over the malleoli and the head of the fibula, as it has been noted that these are the most common locations for pressure symptoms to develop. A Steinmann pin was put through the lower end of the right tibia in the usual manner, a few hours after her admission to the hospital. This was three days after the accident had occurred.

The patient is still in the cast seven weeks after the accident, able to be moved about,

*See Anderson,[4] Figure 10.

sitting propped up in bed, and in every respect comfortable and easily cared for by the nurses.

Case II. J. P., male, aged thirteen, weight 90 pounds.

Diagnosis: Complete fracture of middle third of the left femur with approximately 2 inches shortening (Fig. 11).

The patient was injured by a fall from a horse and he was unconscious for a few moments, after which he was unable to move his left leg.

Twenty cubic centimeters of 2 per cent novocaine were injected into the fracture site. Another small amount of novocaine was injected into the lower end of the left femur and a Steinmann pin was inserted at this point. The well-leg countertraction splint was then applied. The casts on both the well and injured leg extended to the upper thigh. The splint, pin and casts were removed at the end of five weeks. He was seen for the last time at the end of two months and was bearing his weight on the extremity. There was no apparent or real shortening and the functional results were excellent.

Case III. J. S., male aged thirty-six.

Diagnosis: Complete fracture of the lower third of the left femur with the distal fragment displaced posteriorly. Plates of the pelvis showed what appeared to be an old bilateral deformity of the femoral necks due to Perthes' disease (Fig. 10).

The patient was struck by an automobile. He complained of pain and inability to move the left thigh and also of some pain in both hips.

Under local anesthesia, the well-leg countertraction splint (Fig. 7) was applied. The cast was first put on the right leg up to the middle of the thigh. A Steinmann pin was put through the lower end of the left tibia and another pin inserted through the left femur above the condyles. The purpose of the pin through the femur was to overcome the pull of the gastrocnemius muscle, not possible by direct extension when the fracture is in the lower third of the shaft of the femur.

The technique has been altered in the last few cases only one pin being used through the lower end of the femur and none through the tibia. Traction was exerted by attaching the leg to the Hawley table and anterior traction was made on the pin. A cast was applied from the ankle to the upper portion of the thigh after reduction of the fracture and after it had set, the cast was continued down over the toes and the well-leg countertraction splint applied. This method necessitates the use of only one pin.

Both pins, the splint and casts will be removed at the same time after about nine weeks of immobilization.

Case IV. L. K., negress, aged forty-nine.

Diagnosis: Comminuted supracondylar T-fracture of the left femur extending into the knee joint (Fig. 11).

The patient was riding on the back seat of an automobile when the machine struck something and she was thrown out, injuring her left leg.

Treatment was instituted by putting the patient on a Hawley table and applying traction to the left leg. A Steinmann pin with adjustable sleeves (Fig. 5, B) was inserted through the distal fragments of the fractured femur and lateral pressure was added to traction to effect reduction. A unilateral hip spica was then applied from the toes to the chest, incorporating the pin in the cast to fix the fragments after reduction.

Case V. A. Mc., female, aged forty-six.

Diagnosis: Comminuted fracture of the middle third of left tibia and fibula. (Fig. 12).

The patient fell from a ladder a distance of 12 feet.

Under nitrous oxide anesthesia, a Steinmann pin was inserted through both the upper and lower ends of the left tibia. The lateral adjustable metal splints (Fig. 6) were then applied. Reduction was checked by *x*-rays and the cast applied from the toes to the mid thigh, incorporating both pins. The position was checked by subsequent roentgenograms and the patient permitted to return home.

The pins and cast were removed at the end of nine weeks. The functional and anatomical results were excellent.

The use of these splints is similar to the method suggested by Abbott in his operative lengthening of the tibia. Pitkin and Blackfield also reported a series of 12 cases in which a similar treatment was instituted.

Case VI. E. S., female, aged seventy-four years.

Diagnosis: Comminuted fracture of middle third of the right humerus (Fig. 13).

The patient was in an automobile accident in which the car turned over, injuring her right arm.

She was an obese female of short stature and attempts at closed reduction with cast application failed to get good alignment on two attempts.

Ten days later, under nitrous oxide anesthesia, an open reduction was done. The bone ends were approximated and held in position by braided wire. The wound was closed. Because of the earlier difficulty encountered in holding the arm in satisfactory position while applying the cast, a divided Steinmann pin was put through the olecranon process of the right ulna. Traction was maintained through the medium of this pin, which was embedded in the cast. Nine weeks later the cast was removed. Following physiotherapy, the function in the arm returned quite satisfactorily. Flexion was about 75 per cent of normal.

The divided pin broke in this case and this experience has militated against its future employment in our hands, for reasons given earlier in the paper.

SUMMARY

In the foregoing case reports of fractures in the long bones where traction was needed, the Steinmann pin method was used. This has been so satisfactory in our hands that change to another method is deemed unnecessary. The cost is minimal as there is no expensive equipment to purchase. A pin may be put in place with instruments requiring an outlay of but a few dollars, and subsequently only a few cents for each pin.

There has been no instance of osteomyelitis developing in our series of about 70 cases in which pin traction was employed. Roger Anderson has not had a bone infection during a five-year period in which he used the pin for skeletal traction. Should a draining sinus persist, it may be easily curretted, as was suggested by Conwell who uses a pin in severe compound fractures in patients engaged in coal and iron mine work. By the use of strict surgical asepsis, there is reasonable assurance that the wound will not become infected. We have never employed preliminary drilling, as has been the custom in some localities.

The popularity of the piano wire type of traction and the feeling that infection is less likely to occur are due in a measure to the sense of security existing with a small hole in the bone as contrasted with a slightly larger one produced by the pin. It would seem reasonable to assume that there was no greater danger in the larger diameter of wire for skeletal traction, than in a larger incision for abdominal exploration, for infection may occur in either instance, due to faulty technique. Emphasis must be placed, however, on avoiding the changing of dressings around the pin. Care must be exercised also in removing the pin and frequently one will note a small amount of serum following the withdrawal of the pin. The serum has been cultured and found to be sterile and subsequent complete healing of the wound has confirmed the bacteriological finding.

The dangers of slipping and cutting the bone by the wire are as great as, or greater than, in the use of the pin. Burkle reports several cases in which the wire did cut the bone and result in a suppurative arthritis requiring amputation.

Plaster-embedded skeletal traction as advocated by Orr and Thomson, and Speed, which we have used almost exclusively, practically precludes the complication of skeletal traction slipping and cutting the bone. Fixed traction also minimizes the percentage of non-union of fractures.

It has been held that traction through a joint weakens its ligaments, and if disproportionate force is exerted on the ligaments of a patient who has been weakened by muscular disuse and sepsis a joint injury is likely to result. Judgment and experience will obviate employment of traction through a joint when the surgeon finds these contraindications to exist. Hey Groves feels that ligament stretching is practically negligible, but on the other hand distraction of the joint surfaces produced by extension affords a valuable

factor in maintaining the mobility of the joint, particularly so in the elbow and ankle. When there is a fracture near any joint, stiffness of the articulation is a great danger to be feared and guarded against. On the other hand flail joints are unknown following traction through a joint, except in paralytic conditions or cases where a large amount of bone has been lost. Physiotherapy after the fracture has healed readily overcomes joint stiffness from disuse.

Pain at the site of the pin is a rare complication. Usually the patient is not aware of the presence of the pin unless told about it. Reduction of swelling and the resulting looseness of the pin in the cast may cause pain unless the cast is changed and rigid fixation maintained by means of a new cast.

CONCLUSIONS

Skeletal traction with Steinmann pins is a most reliable and efficient method for treating fractures.

Embedding the pins in plaster casts, as we have done in all of our cases, makes for a true fixed dressing with immobilization in correct position as an accomplished fact.

Rigid fixation of pins in plaster is less provocative of bone cutting and infection than constantly shifting of pins when weights, ropes and pulleys are used.

The undivided or solid Steinmann pin is empirically safe. Five years in private practice and two years' experience on a county hospital service, where the responsibilities of the house staff are greater, and with only a single incidence of infection, either before or after removal of the pin, are proofs against adverse criticism.

The modified Steinmann pin with adjustable sleeves for lateral pressure and the special clamps for the same purpose are of great value in reducing and maintaining the position of laterally displaced bone fragments, especially when the fracture extends into a joint.

The well-leg countertraction splint employing skeletal traction for the injured leg is an original splint devised by Roger Anderson. The splint has been used successfully for dislocation of the pubic bone, for injuries and fractures of the pelvis, for fractures of the neck and shaft of the femur, and for fractures of the tibia.

My appreciation is due Dr. Donald V. Trueblood, who sponsored this thesis as my preceptor.

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[For Remainder of References see p. 477.]

MULTIPLE OSSIFICATION CENTERS OF THE PATELLA*

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WINNETKA, ILL.

ALTHOUGH the error of making the diagnosis of fracture in multipartite patellae was more common before the days of Roentgen, nevertheless the confusion still exists in many instances.

On account of its development and anatomical location, the patella is usually considered a sesamoid bone.[1] Commonly it ossifies from a single center in the second or third year of life though occasionally not until the fifth or sixth. These centers normally close by the age of twelve or fourteen. However, one may find two, three, or even more centers of ossification and in such instances these separate parts may never fuse, and are then held together by a fibrous union. The earliest description of this condition is that made by Wenzel Gruber[2] of Petrograd in 1883. He believed that owing to inhibition of growth processes, these centers fail to fuse into a single patella. Following this author there were several brief accounts in the literature until 1921, when Dr. Sindling-Larsen[3] recorded what he believed to be a new clinical entity.

In two otherwise healthy girls of ten and eleven years, respectively, who after overstraining themselves by dancing and jumping had complained of pains in their knees. When the patients were seen by the author, the affection was in each case unilateral; the pole of affected patella was painful on pressure and in one of the cases the soft parts over and below the patella were slightly swollen. In the roentgenograms of the knees taken in profile, the anterior and lower outlines of the painful patellae were hazy with abnormal calcium deposits or bone shadows in the periosteum below the patellar apices.

The author interpreted these findings as indicating epiphysitis caused by overstrain, and offers in support of his conclusions the finding of a mild Schlatter's disease of the tibial apophysis in one case. The roentgenograms of the healthy knee in both cases showed similar abnormalities in a lesser degree. The first case was treated by plaster immobilization for six weeks; the second, with rest without fixation. In each case a complete recovery both clinically and roentgenologically resulted after six months treatment.

In 1922, Dr. Sven Johansson,[4] unaware of Larsen's paper, reported the same condition. He records four cases. Each of these patients made a prompt recovery after the knee had been relieved from strain for a month or two.

Kempson[12] in 1902 pointed out that the usual accessory center of ossification occurs at the outer margin of the patella. In 1921, McNally,[13] in a study of a large series of patellae taken from cadavers, found that 3 per cent had a more or less marked defect at the upper outer margin of the patella with or without a true accessory center of ossification at this site. He found that such defects were bilateral in two-thirds and unilateral in one-third of the cases. Of special interest was one with a definite partially fused separate center of ossification at the attachment of the patellar ligament to its lower pole.

Hawley and Griswold[5] believe that both Larsen's and Schlatter's disease are essentially traumatic, resulting from a strain of the patellar ligament which is in itself potentially strong, but in both Osgood-Schlatter's a nd Larsen-Johansson's disease, is attached below or above into a weak point of anchorage.

Buttner[6] describes a case of Perthe's disease and tripartite patella in a girl of eleven years. The child was born with bilateral club feet. There was no evidence of rickets. She learned to walk at the age of one. About one year previous to the report pain in both hips and knee joints developed without fever. After a failure

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with medical treatment the patient was admitted to the hospital. The blood Wassermann reaction was negative. There



Fig. 1.



Fig. 2.

was a moderate lymphocytosis. Both thighs flexed to about 40 degrees at the hip joints and there was a corresponding lordosis of the spine, especially in the lower dorsal portion. The trochanters were elevated on both sides. The knee joints were thick but movable. There was an abnormal amount of lateral mobility, especially on the right. *X*-ray examination of Buttner's case revealed a bilateral Perthe's disease of the hip joints. The roentgenograms also revealed multiple deformities of the skeletal system. He states that the peculiar condition of the patella, in which three ossification centers were seen, could not be found referred to anywhere in the literature (1925).

Fleischner reports a unilateral case with a Schlatter's disease of the other side.

Odermott[8] believes that lateral accessory ossifications occur along the patella proper. It is uncertain whether an ontogenetic triple division can also be assumed. The regularity and similar shape of the three parts would seem to support such a theory. His case presented a manifold complication of skeletal deformities, including:

1. Rarefactions of the skull
2. Flattening of the vertebrae
3. Flattened epiphyses
4. Joint surfaces of the elbow flattened
5. Flattened epiphyses of the hand joints
6. Marked flattening of the distal epiphyses of the metacarpals
7. Flattening of the epiphyses of the femoral head
8. In both knee-joints, the epiphyses of the femur and tibia were flattened
9. The patellae were tripartite on both sides
10. Bilateral flattening of the lower tibial epiphyses
11. Bilateral bone atrophy at the distal end of the first metatarsals. With such an

array of pathology in one case, there is ample need for further investigation of epiphyseal disease from the standpoint of constitution, heredity and degenerative stigmata.

Reinbold[7] reports a case of unilateral tripartite patella in a man of twenty-four years. He twisted his left knee in descending the stairs and developed a hydrarthrosis. *X*-ray examination revealed three centers of ossification in the patella detached from one another. The patient gradually recovered the full use of his joint and the patella condition was therefore not attributed to the slight trauma.

Odermott[8] distinguishes three anomalous types of patella:

1. Division by a horizontal cleft in the lower third or fourth
2. Lateral development of one or more accessory fragments
3. The most frequent development is in the upper lateral quadrant of one or more accessory fragments.

Odermott[8] describes another case in a man of fifty-eight, who had suffered a fall on his right knee. Roentgenograms showed several fragments, partly with smooth opposing surfaces in the upper quadrant of the patella. The fragments completed the normal shape of the patella. The left patella was normal. At operation two larger and several smaller bones were found. The opposing surfaces of the larger fragments were smooth. In the smaller fragments, roentgen examination revealed rarefactions which histologically proved to be genuine bone marrow spaces. From the histologic study, it seemed probable that this was a case of non-fusion of multiple osseous nuclei. In other words an anomalous development of the patella.

I report the following case:

Mr. W. L. aged fifty-five years, entered the Northwestern University Medical School Surgical Dispensary on February 25, 1931. He was complaining of a swollen, red, tender area, about 4 inches in diameter, on the anterior aspect of the right leg. This was obviously a cellulitis, which on further questioning was found to have followed the scratching of a louse bite, with which he was copiously infested. In making a complete examination, it was noted that each knee was peculiarly deformed, although there was no loss of motion or subjective weakness in either. He stated that he had had each knee operated upon "because the knee caps were pulled apart." No other reason for operation could be elicited from the patient.

The right knee had been operated upon in July, 1907, at which time the fragments were sutured together with catgut. A posterior extension of splint was worn for one month. The segments of the patella again pulled apart after one year.

The left knee was operated upon at another hospital in February, 1912. This time steel wire was used to suture the fragments, after which a posterior retention splint was worn for two weeks. The fragments of the left patella at the time of this examination, although somewhat close in apposition, were readily moved apart and laterally on palpation.

Owing to the intervening length of time since the operations, the previous hospital records had been destroyed, so it was consequently impossible to procure a more accurate check on this case.

The *x*-ray film shows two large and two small fragments of the left patella. The right shows a main piece, consisting of perhaps three-fifths or two-thirds of the patella, riding high up above the lower end of the femur. The rest of the patella is found in two pieces. The separation is much more marked in the right knee. There is also seen a fabella in the long head of the gastrocnemius on the left side. (Dr. James T. Case.)

Of the recorded cases, one finds multiple centers of ossification in about 15 per cent of the number. These do not necessarily mean a divided patella however, as they may fuse to form a normal patella. Among 410 knee roentgenograms, Odermott found 15 bipartite or multipartite patellae.

The deformity may give rise to no symptoms, or by fracture of the parts lead to arthritic symptoms. The soft tissues separating the fragments may become injured, stretched or torn, leading to swelling and pain of the knee joint, and eventually to a false movability of the

fragments in relation to each other. The distortion may heal or lead to symptoms resembling a chronic arthritis. The usual therapy for joint effusions or arthritis is indicated in such cases.

Operative removal of the fragments is indicated only in case of chronic irritation, and suturing of the larger pieces together only if there is a weakness of the knee joint. This latter is usually one of inability to completely extend the leg.

In compensation cases, these injuries play a significant part and must be differentiated from fracture.

Inasmuch as this condition seems analogous to Schlatter's disease of the tibia, it should, when recognized in early adolescent life, be treated so as to keep as much strain as possible away from the patella. By this means the separate centers may thereby have some chance of fusing together.

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SURGERY OF THE SYMPATHETIC NERVOUS SYSTEM

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THE recent advances in surgery of the sympathetic nervous system have stimulated widespread interest because certain types of disease, hitherto resistant to other forms of treatment, have been relieved. It is the purpose of this paper to present the type of disease condition which will favorably respond to sympathectomy and to outline briefly the method of selection of cases and the operative procedures.

HISTORICAL SURVEY

The first sympathetic ganglionectomy was performed by Alexander in 1899 when he removed both superior cervical ganglia for epilepsy; in 1896 Jonesco divided the trunk above and below the middle cervical ganglion for epilepsy and a year later repeated this operation for exophthalmic goiter. Franck's discussion in 1898 lent new interest to the sympathetic system when he considered that angina pectoris was the result of an overflow of painful impulses reaching the cord via the stellate ganglion. Over thirty years later Ransom corroborated Franck's view by stating that sensory fibers carry impulses to the spinal cord through the white rami.

In 1899 Jaboulay cut the nerves approaching an artery and Higier, 1901, recommended tearing the nerve plexus around the femoral artery in cases of intermittent claudication. Leriche, who is credited with the establishment of periarterial sympathectomy, reported two cases of causalgia in median nerve injuries in which he resected the sheath of the brachial artery in 1917. Since then he has reported large series of cases with favorable results lasting from a few months to several years. Leriche advocates periarterial sheath resection in all cases of vasospastic disorders, such as Raynaud's disease, causalgia, trophic ulcers and the superinduced spasm of the collateral vessels in thromboangiitis obliterans, as he believes there is an intramural ganglion in the arterial wall. However Kramer and Todd and Potts and Kuntz have demonstrated that the sympathetic innervation of the peripheral vessels corresponds to the musculocutaneous distribution of the spinal nerves.

Hunter and Royle, 1924, reported beneficial results from ramisectomy in cases of spastic paraplegia, and at that time noted the extremities operated upon were warm, dry and pink. Adson and Brown, utilizing this knowledge, applied it to Raynaud's disease, and in 1924 reported the first cases of lumbar sympathetic ganglionectomy and ramisectomy with brilliant results. Adson prefers the transabdominal route because it is possible to see the inconstant rami emerging from the first lumbar ganglion, which would be missed by the retroperitoneal approach.

The anterior approach to the stellate ganglion for vasospastic conditions in the upper extremities was not very successful until in 1928 Adson devised the posterior approach following the suggestion of Henry.

In the past decade a number of surgeons have utilized either sympathectomy or alcohol injection of the sympathetic ganglia for various types of intractable pain. The work of Swetlow, White and Mixter in relieving the pain of angina pectoris, Archibald and Scrimger and Verbrugge and von Bogaert in alleviating abdominal pain and Stern in relieving the distress of thromboangiitis obliterans has been particularly outstanding. Later Adson, Rowntree and Henderson reported marked benefit in certain cases of arthritis when sympathectomy was performed. More recently Flothow and Abbott have reported cases of

intractible pain in various regions of the body which have been alleviated by alcohol injection of the sympathetic nervous system.

DISEASES AMENABLE TO SYMPATHECTOMY

Raynaud's Disease. This is a classic vasospastic condition and is characterized by a bilateral symmetrical vasoconstriction of the hands and feet. Little has been added to Raynaud's original description when he observed that there is a form of gangrene of the extremities without any evidence of vascular occlusion. This phenomenon varies in degree to such an extent that many cases of mild vasoconstriction are confused with Raynaud's disease. These patients complain of a coldness of the hands and feet, some blanching of one or more digits and a moderate amount of pain when exposed to the cold. Although there is no contraindication to surgery in these cases, it seems a drastic procedure when relief may be obtained by the application of external heat, massage and wearing warmer clothes.

True Raynaud's disease is characterized by the presence of symmetric color changes in the extremities, either upper or lower. The knees, elbows, ears and tip of the nose may also be involved in severe cases. The blanching of the skin is induced by exposure to the cold or emotional influences, and this is followed by cyanosis until the vasospasm subsides. The gangrene of Raynaud's disease is dry and occurs at the tips of the fingers and toes with distortion of the growth of the nails, instead of complete gangrene of a digit as seen in occlusive vascular disease.

Raynaud's disease is chiefly seen in women and few cases in men prove to be a pure vasoconstriction without at least some changes in the lumen of the artery. The surgical procedure carries no more risk than a laparotomy and the relief from pain is well worth that small risk. Immediately after operation the fingers or toes are free from pain and are warm, dry and pink. The trophic lesions soon heal and the nails rare restored to normal growth. The Horner's syndrome following cervico-thoracic sympathectomy consists of a constriction of the pupil, narrowing of the palpebral fissures and an absence of perspiration over the face. The retinal vessels are dilated and the caliber of the capillaries of the mucous membrane of the nose is enlarged so that there is an increase of mucous secretion. The skin is usually quite dry after operation but the application of cold cream or cocoa butter is sufficient to relieve this slight discomfort which is nothing in comparison to the previous pain from vasoconstriction.

Scleroderma. This is a disease of the skin characterized by pigmentation, thickening or atrophy, and is frequently associated with sclerodactylia. The distribution is inconstant but is usually confined to the extremities, face, neck and chest, although the entire trunk may be involved in severe cases. Muscle and joint action may be limited in this condition. Not all cases of scleroderma are caused by vasoconstriction but many patients in this group give a history of cold, clammy cyanotic extremities prior to the skin changes. This type of case responds to the sympathetic ganglionectomy as the extremities become warm, dry and pink and there is a gradual loosening of the tight skin.

Causalgia. This is a painful condition following injuries to the peripheral nerves, chiefly the ulnar, median and sciatic nerves, and is characterized by pain, glossy, red or mottled red and white skin and limited joint motion. Sympathectomy or alcohol injection of the sympathetic ganglia has offered relief in a number of cases, however the psychic and compensation element is often a paramount factor in this type of disease.

Arthritis. There is a small group of young adults who have painful, swollen, tender joints, limited motion and cold, clammy extremities. External heat gives a moderate degree of relief and when typhoid vaccine is administered intravenously there is an increase in the temperature of the extremities with a temporary relief from pain. A permanent result is obtained by

sympathectomy but extreme care must be exerted in the selection of cases.

Thromboangiitis Obliterans. This condition is due to an occlusion of one or more of the main arteries to an extremity associated with varying degrees of color changes, pain and extensive gangrene. Buerger's original description stated that this disease occurred only in Hebrews and that tobacco, chiefly cigarette smoking, was a definite contributing factor. However it has since been demonstrated that no race is immune and that although most patients are cigarette smokers, this condition is seen in nonsmokers. The course of this disease is slow and for some time it may be manifested only by intermittent claudication and coldness of the extremities. Later color changes appear which vary from a deep rubor to marked cyanosis which is replaced by blanching when the extremity is elevated for a short time. Gangrene usually begins around the nail and involves all or part of one digit. Later it progresses until amputation is necessary unless adequate collateral circulation can be obtained.

The only means of producing adequate collateral circulation is to relieve the spasm of the vessels which are not occluded. This can be accomplished by severing the sympathetic fibers causing this vasoconstriction. It is obvious that these cases must be carefully selected so that there is an assurance that the collateral circulation will be adequate. Thromboangiitis obliterans occurs chiefly in men and care should be taken to avoid confusing early cases with Raynaud's disease because of a palpable vessel, as Brown and Allen have called attention to errors in diagnosis when pulsations were still present in distal arteries.

Arterial Sclerosis. Little can be hoped for in dilating the vessels of occlusive conditions in the later decades of life, but relief from intractable pain can be accomplished by alcohol injection of the sympathetic ganglia. The risk of injection is almost nil and is preferable to massive doses of narcotics in the hope of affording relief to these unfortunate suffers. It is also a distinct advantage to allay this intense distress so that the patient can be built up for amputation, if necessary, at a later date with a better assurance of healing and the elimination of the painful stump.

SELECTION OF CASES

The success of sympathectomy in the treatment of peripheral vascular disease depends entirely upon two factors, the proper selection of cases and the thorough interruption of all of the vasoconstrictor fibers. There are, at present, four methods of determining the suitable cases: (1) the "fever" test, (2) spinal anesthesia, (3) direct injection of nerve trunks and (4) direct injection of the sympathetic ganglia.

1. *The "fever" test* was devised by Adson and Brown in 1925 and consists of administering 25,000,000 to 75,000,000 dead bacilli in typhoid vaccine intravenously, according to the age of the patient, and estimating the rise in the surface temperature of the extremities in contrast to the rise in mouth temperature. This is an accurate means of determining the relative increase of blood flow in the vessels of the extremities. The objection to this method is that it causes a severe generalized reaction and is an indirect test because it only produces vasodilatation without giving information as to relief of pain.

2. *Spinal anesthesia* will produce vasodilatation but this method is limited because it gives no indication as to pain relief, as all sensations are absent, and is practical only for the lower extremities.

3. *Direct injection of the nerve trunks* often provokes technical difficulties and is incomplete as all sympathetic fibers are not interrupted.

4. *Direct injection of the sympathetic ganglia,* as advocated by White, produces a selective and discriminative result as both vasodilatation and pain relief are accomplished in as limited an area as desired.

In all of the foregoing diagnostic methods, it is essential to secure sufficient

vasodilatation or pain relief to warrant surgical procedures on sympathetic ganglia and rami.

Pain of Head, Face and Neck. Mixter and White postulated the theory that some of the pain fibers from the head and neck coursed through the inferior cervical and first thoracic sympathetic ganglia on their way to a central point of perception. This has been corroborated by Flothow who removed the stellate and second thoracic sympathetic ganglia in a case of facial neuralgia after section of the postganglionic root of the trigeminal nerve had failed to afford relief, and Abbott reported a similar case relieved by alcohol injection of the same ganglia. Dandy has removed the stellate and second thoracic ganglia for hemicrania and although the patients have not been observed over a very long period the results have been satisfactory to date. On the other hand, Frazier was disappointed in his cases in which he removed the cervical sympathetic chain by the anterior approach. However his results might have been more satisfactory if the posterior approach of Adson had been used. There is no doubt but that certain painful conditions of the head and neck can be relieved by cervicothoracic sympathectomy or alcohol injection when other methods fail.

Angina Pectoris. Many surgical procedures on the sympathetic nervous system have been devised for the relief of the pain of angina pectoris since Franck's original suggestion in 1899. It is universally agreed that any surgical procedure in this condition is only palliative but relief of pain is important not only from the standpoint of the patient but, as suggested by Wenckebach and Danielopolu, the pain in itself may be actually injurious to the heart.

The knowledge of the innervation of the heart is incomplete but it is generally conceded that the upper sympathetic ganglia are definitely involved so that ablation of these ganglia seems a logical procedure.

The most plausible theory of angina pectoris is that advocated by Danielopolu, which is based on the assumption of an inadequate coronary circulation whether there is a coronary sclerosis or an apparently normal heart. Thus the anginal attack arises from a disturbance of the balance between the work of the myocardium and the blood supply. The various operations consist of resection of different portions of the upper sympathetic ganglia and the most complete procedure is that of Jonesco, which is a resection of the entire cervical chain and first thoracic ganglia. Other operations consist of removal of the superior cervical ganglia, right, left or both, section of the trunk below the superior cervical ganglion and section of the superior cardiac nerve, removal of the superior and middle cervical ganglia, and removal of the stellate and second thoracic ganglia. No one type of operation has been universally successful. In 1924 Brunn and Mandl tried paravertebral nerve block with procaine with splendid results. Later Swetlow began the use of alcohol in these injections and since his original work many others have used this method with more constant results than following the operative treatment. This procedure seems most logical as it interrupts the visceral afferent pain fibers which pass through the rami communicantes and dorsal roots regardless of the route up to that point.

White injects the upper five thoracic roots with 5 c.c. of 1 per cent procaine followed by 5 c.c. of 85 per cent alcohol. This seems to be the most efficient method as a large percentage of the patients obtain immediate relief from pain which lasts for several months.

The only deleterious effects of the injection are disagreeable paresthesias in the segmental distribution but these last only a short time and are not as severe as the previous cardiac pain. There is no operative mortality in skilled hands. This seems a most satisfactory procedure because all patients suffering from anginal

pain are not good risks for as formidable an operation as sympathetic ganglionectomy.

Asthma. The reports of the surgical procedures on the thoracic sympathetic ganglia are so conflicting that, at present, no definite opinion can be formed in reference to the advisability of operation or alcohol injection for asthma.

Abdominal Pain. There is no problem more perplexing than that of a patient with a severe and often disabling abdominal pain which has failed to respond to the usual series of operations on the stomach, gall bladder, appendix or pelvic organs. There is a group of patients in whom the neurogenic factor is of paramount importance. In addition there is a group of patients suffering from inoperable neoplasms, which should be afforded relief from pain without resorting to massive and increasing doses of narcotics.

In recent years several means of interrupting pathways of pain have been devised with varying degrees of success. However it is now established that all of the visceral fibers in the sympathetic nervous system are connected with the posterior root ganglia of the spinal cord.

The earlier efforts to relieve intractable pain in the abdomen were confined to section of the posterior roots and cordotomy but too often these measures did not afford freedom from suffering. Later Von Gaza advocated section of the rami communicates and von Bogaert and Verbrugge achieved success in allaying various types of abdominal distress, including tabetic crisis, by this operation. Woodbridge has obtained satisfactory results by alcohol injections of the sympathetic ganglia and the results in the author's experience have been equally gratifying.

The distress of idiopathic dilatation of the colon or Hirschsprung's disease has been solved by lumbar sympathetic ganglionectomy as demonstrated by the reports of Wade and Royle, Judd and Adson, Rankin and Learmonth and Robertson. Many painful conditions in the pelvis which were unexplained by demonstrable pathology, or the lack of it, have been relieved by section of the hypogastric plexus or presacral nerve as indicated by the work of Leriche, Hovelaque, Fontaine and Herman. Learmonth's result in alleviating the distress of cord bladder and painful conditions of the lower urinary tract has solved a long-felt want and has been an outstanding contribution.

Although many advances have been made in relieving severe, and often unexplained, abdominal pain by section or injection of the sympathetic nervous system, much is to be learned by the possibilities, limitations and disadvantages of such procedures. It is obvious that extreme caution must be exercised in the selection of patients for such operations with due regard for the psychic element in each individual case.

OPERATIVE PROCEDURES

1. *Periarterial sympathectomy* consists of stripping the adventitia of the artery for a distance of 2 or 3 inches. This is facilitated by a preliminary injection of the adventitia with physiological saline solution.

2. *Cervicothoracic sympathetic ganglionectomy* is performed by making a midline incision from the tip of the spinous process of the fifth cervical vertebra to the fourth thoracic vertebra. The tendinous attachments of the trapezius, rhomboid and posterior serratus muscles are divided and the erector spinae and lower end of the splenius capitus muscles are exposed. A blunt dissection is made over the first thoracic vertebra and then a subperiosteal resection of 3 cm. of the rib and transverse process of the first thoracic vertebra is effected. The lung and pleura are gently dissected from the vertebra and retracted laterally. The sympathetic trunk is exposed and the second thoracic ganglion and rami are divided; then traction downward is made and the stellate ganglion, with its rami, is removed.

3. *Lumbar sympathetic ganglionectomy.* The patient is placed in the Trendelenburg

position. A midline abdominal incision is then made and the intestines pushed away. Adson mobilizes the sigmoid and lower portion of the descending colon by incising the parietal peritoneum laterally. Then the colon and ureter are retracted to the midline, also the aorta and vena cava are retracted mesially. The sympathetic trunk is removed from below the fourth lumbar ganglion up to, and including, the second lumbar ganglion and all the rami. On the right side, the incision in the peritoneum is made just lateral to the aorta and vena cava. The cecum, small intestine, ureter and the psoas muscles are then retracted laterally and the great vessels medially. Removal of the sympathetic trunk is similar to that on the left side.

An incision in the peritoneum immediately below the bifurcation of the aorta and carried upward above the aorta will facilitate exposure and diminish operating time.

Resection of the hypogastric plexus or presacral nerve is accomplished by incising the peritoneum below the bifurcation of the aorta and retracting the two sides of peritoneum laterally. The pelvic portion of the sympathetic chain is then removed.

SUMMARY

A brief review of the recent advances in the surgery of the sympathetic nervous system is presented in the hope that further interest will be aroused that will tend to a more complete knowledge of this mechanism, which undoubtedly holds the key to many unsolved problems. In such a limited survey of this field many valuable references have been omitted and it is with regret that all worthwhile contributions cannot be listed. However it is obvious that careful and deliberate selection of patient and operation will be of distinct advantage in many apparently hopeless cases, with a minimal mortality in skilled hands.

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RECTAL ADMINISTRATION OF TRIBROMETHANOL

ITS USE ABROAD*

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NEW HAVEN, CONN.

THE chemical compound tribromethanol was produced by Willstaetter, the Nobel prize winner of 1915, and Duisberg[143] and has been in use as a general anesthetic on the Continent since 1927. However, reports regarding the value of the drug, its action, indications for its use, dosage, method of administration, etc. are at great variance (Anschuetz;[1,2,3] Boeters;[11] M. Borchardt;[13] Christ;[19] Domanig;[25] Heuss;[58] E. Muehsam;[88] R. Mühsam;[89] Sauerbruch[111]). It seemed desirable therefore to assemble the available data and thus gain an idea of the true situation (Goldschmidt and Harvey[48]). The procedure adopted was that of reviewing all of the literature abroad and of collecting information through direct inquiry by correspondence with the authors and heads of the respective institutions regarding their experiences and opinions on tribromethanol anesthesia.

The results of these studies indicate that failures and accidents may be justly ascribed to unskilled application of the anesthetic and that by proper indication, proper dosage and proper method of administration of tribromethanol these may be easily avoided and furthermore that the introduction of this drug as a basal anesthetic marks a new era in modern anesthesia.

PHYSICAL, CHEMICAL AND PHARMACOLOGICAL PROPERTIES

Tribromethanol (E 107, avertin, Byck 250, renarcol) is a white, crystalline substance, soluble in water at a temperature of 40° c. Chemically it is a tribromethyl alcohol, the result of an only vaguely described patented chemical process (S. Loewe;[80] Straub;[130] Herzberg;[57] Nestmann;[93] Welsch[139]). Formerly it has been given orally (Eckstein). At present, the intravenous (Kirschner,[64] Stich[129]) but mostly the rectal administrations are in use (Sebening[120]). According to the manufacturers' instructions, avertin fluid should be given in a 2½ per cent solution, freshly dissolved at body temperature in doses of 0.08 – 0.1 c.c. per kilogram of body weight. This dose should be lowered when the drug is given to debilitated, (Kleesattel;[65] Guleke[51]) cachectic, dehydrated or obese patients (Specht[125]) and raised for children and strong individuals between the ages of twenty and thirty-five years. In practice a variety of criteria are used for determining the dosage, such as age (B. Martin[81]), purpose of administration, whether basic or general anesthesia (B. Martin;[81,82] Sievers;[122] Kirschner;[64] Stich[129]) is desired. The action of the drugb yrectal administration, which is now the generally accepted method, depends on a number of factors (Roedelius[109]). One of these is the degree of absorption which varies according to the concentration of the solution used (Straub;[130] Kotzoglu;[68] Mörl[85]) and also on certain physical conditions such as the pressure under which the enema is introduced, position of the patient (Anschütz;[1] Goecke;[46] Haack;[52] Treplin[133]), etc. Another factor is the method of preoperative preparation of the patient. The combined use of narcotics and of admixture

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(Fränkel;[38] Riedel[108]) of various drugs in the tribromethanol enema modify its action (Grossmann[50]). It acts differently in man than in woman, the latter being more sensitive (Beck[7a]; Beck and Lendle;[7,60,73,74] Bolz and Borchers;[12] Cauchard and Monod;[18] Endoh;[32] Kaczander;[59] Kärber[60]). In animals the lethal dose is constant, 1.75 times higher than the full anesthetic dose (Anschütz[1]). The exact routes of the excretion of tribromethanol are not known (Wymer[146,147] and Fuss). Most of it is believed to be eliminated through the urine. According to Nordmann[95] the greater bulk is excreted during the first six hours but traces remain in the urine after two days. According to Sebening[120] and Straub[130] when 15 gr. of a 3 per cent solution of tribromethanol are administered, half of the drug is absorbed in ten minutes, three-fourths of it in twenty minutes. The rest is so slowly removed that 5 or 10 per cent is present in the fluid returned two hours after the instillation.

Of the pharmacological actions of tribromethanol which justify its preferred use to other anesthetics the following may be mentioned. The stage of excitement is absent, the patient gradually loses consciousness within two to fifteen minutes, surgical anesthesia is obtained after thirty minutes lasting for about two hours and is followed by sleep and analgesia for about four hours. There is complete amnesia of all events while under the action of the drug. Nausea and vomiting as after effects of the anesthesia are less severe or absent. A drop of the blood pressure without change in the pulse rate is observed during the instillation of the anesthetic. This, however, is no cause for alarm since it quickly returns to the level observed in normal sleep (Church;[20] Goldschmidt and Harvey;[48] Boas and Goldschmidt;[10] Kennedy;[62] Stuber;[131] Unger and May;[136,137] Wessely[140]).

Excitement after the anesthesia has been occasionally observed. According to Lexer,[75] Lobenhoffer[77] and Schulze[119] alcoholism may be responsible for it.

METHODS OF TRIBROMETHANOL INSTILLATION

In reviewing the literature and the answers to our inquiries, it was learned that there is a group of clinics which do not use tribromethanol at all (Table I), one which uses the drug for basal anesthesia and follows exactly the instructions of the manufacturer (Table II), and a third group which applies tribromethanol rectally but by individual methods (Table III). Here we are particularly concerned with the latter group.

1. *Nordmann's Method.*[95] Nordmann formerly used avertin in an enema which contained a demulcent of salep.* Later he found that this was not helpful and discontinued its use. Subsequently he added to the enema magnesium sulphate and calcium chloride but soon dispensed with all additions. Nordmann's followers use chloraethyl or solaesthin† as a supplementary anesthetic.

2. *Kreuter's Method.*[69] Kreuter administers tribromethanol in a 3 per cent solution in doses of 0.125 to 0.150 gm. per kilogram of body weight. No laxative is given the evening before the operation but a cleansing enema is given.

3. *Sievers's Method for Children.*[122] Sievers desires to obtain in each case full anesthesia and administers therefore .15 gm. of tribromethanol per kilogram of body weight in a 3 per cent solution. If after ten minutes the patient reacts to cold or pain, an additional dose of .025 gm. of the drug per kilogram of body weight is given. If, after this, complete anesthesia is not obtained, he either cancels the operation or orders a colonic irrigation and repeats the procedure after one hour. (Ebhardt.[27])

4. *B. Martin's Method.*[81] Martin aims to attain full anesthesia with tribromethanol by combining it with other drugs. The

* Demulcent of a dried tuber of a special kind of Orchis growing in Turkey.

† Solaesthin is a methylenchloride, related in its chemical structure to chloroform. It is non-explosive, can be dropped like ether, and is cheaper than chloraethyl.

essentials of this method are the addition of a 25 to 30 ccm. of a 20 per cent solution of magnesium sulphate to the 2½ per cent liquid avertin "stem-solution." He further adds 1 c.c. of a 3 per cent solution of narcophin.* This is omitted in children under fourteen years. The table used for the doses is given below.

Age	Avertin (Gm.)	Magnesium Sulfuricum (Ccm.)	Narcophin
1–14	0.17–0.18	30	
15–24	.15– .17	30	1.0
25–34	.14– .15	30	1.0
35–60	.13– .15	30	1.0
over 60	.13– .15 or less	30	1.0

According to B. Martin[81] the patient is asleep in two minutes, is unconscious after five minutes, and is in deep anesthesia after fifteen minutes. This stage lasts for seventy to one-hundred and ten minutes. He claims that the decrease in the blood pressure is less than by any of the other methods. Some are using Martin's method with the modification of not intending to induce full anesthesia and using therefore less tribromethanol (R. Mühsam,[89] Dax,[23] v. Redwitz[105]). The magnesium sulphate is given intramuscularly by v. Redwitz.[105]

5. *Method of the University of Leipzig* (Payr,[96] Plettner[100]). Laxative is not given the evening before the operation. The patient takes two tablets of adalin. On the morning of the operation again two tablets of adalin are given one hour before the operation, and .01 to .02 gram pantopon is injected hypodermically (none to children). Simultaneously with the instillation of the avertin, diluted in salep, one ampule of cardiazol† is given subcutaneously. If the patient is not in deep sleep after ten minutes a supplementary enema of .025 gm. of avertin per kilogram of body weight is given. The operation is followed by a cleansing enema with camillosan.‡

* Narkotin-morphin-mekonat is a double salt of morphin and narkotin. It is a quite ineffective alkaloid compound with meconic acid.

† Lobelin 0.003 gm. and ephetonin .005 gm.

‡ An infusion of Camomilla Romana or Anthemis nobilis.

TECHNICAL DETAILS OF TRIBROMETHANOL ANESTHESIA AS FOLLOWED IN THE EUROPEAN CLINICS

(a) *Preparation of the lower colon on the evening before the operation* is regarded necessary. Many emphasize that they would rather have too little than too much done. Nordmann,[95] and B. Martin[81] claim to have satisfactory results without any previous preparation of the colon.*

(b) *The administration of a hypnotic the evening before operation* is recommended by the manufacturer[5] who suggests phanodorn, luminal and veronal. Other drugs used are medinal, noctal, veronacetin and veramon (Poppert[101] and Schmieden[116]), dilaudid (Lobenhoffer[77]); adalin (Momburg,[86] Lobenhoffer,[77] R. Mühsam,[89] Payr[96]) narcophin (Seefisch[121]). Opinions differ considerably regarding the necessity and use of hypnotics. Some emphasize their use (Sievers[122]) while others (Glaesmer;[43] B. Martin;[81] v. Eiselsberg;[29] Starlinger,[127] Lexer;[75] Schulze;[119] H. Schmidt;[115] Clairmont[21]) are against it.

(c) *An analgesic before induction of the anesthesia* is administered by many. The manufacturer suggests morphia. Others use narcophin (Seefisch[121]), pantopon (Anschütz;[1] Goetze;[47] Friedrich;[39] Poppert;[101] v. Brandis and Killian;[14] Glahn;[44] Pels-Leusden;[97] Wiedhopf[142]), laudanon (Baum;[6] Anschütz[1]), dilaudid (Nordmann;[95] Wiedhopf[142]), veronal (Clairmont[21]), and narcophin (Stemmler;[128] Seefisch;[121] B. Martin[81]).

(d) *Supplementation of tribromethanol with inhalation anesthetics* (Wuelfing[145]). The following drugs used are given in order of frequency:

1. Ether. It is generally agreed that the quantity of ether necessary is a fraction of that without tribromethanol.

2. Nitrous oxide—Oxygen (Domanig;[25] Flörcken;[36] Mues[90]).

3. Chlorethyl.

* Psychiatrists (Sioli and Neustaudt;[123] Blume;[9] Enke;[33] Westphal;[141] Gallinek;[41] Richet and Joly[107]) used tribromethanol rectally with good results without first preparing the colon.

4. Local anesthesia: novocaine-suprarenin 0.5 per cent solution is used by Lobenhoffer;[77] Sievers;[122] v. Gaza;[42] Mintz.[84] Braun[15] recommends this combination in strumectomies and cholecystectomies only. Läwen,[71] B. Martin[81] and Anschütz[1] are against the use of local anesthesia, fearing the blood pressure change and disturbances in the general condition of the patient.

5. Solaesthin (Nordmann[95] and Mintz[84]).

6. Narcylen (Lobenhoffer[77]).

DISTURBANCES DURING AND AFTER THE AVERTIN ANESTHESIA AND ANTIDOTES FOR THEM

Respiratory difficulties in the beginning, during and after operation have been observed, even when small dosages of the drug have been administered (Pfister[99]). The following antidotes are in use:

1. CO_2—inhalation (Schmieden, Sevening, Pels-Leusden, Anschütz, Heuss, Braun, Martin, Lexer, v. Redwitz, Roedelius, Sudeck, H. Schmidt, Unger, Mintz).

2. Lobelin (Momburg, Baum, Pels-Leusden, Druegg, Schloffer,[114] Moerl, B. Martin, Kreuter).

3. Artificial respiration (v. Eicken, Elsbach).

4. Oxygen (v. Eicken, Elsbach); oxygen alternating with CO_2 (Heile, Rodelius, Sudeck, H. Schmidt).

5. Cardiazol (Lexer, Schulze).

6. Cardiazol-lobelin given subcutaneously the same time as the avertin is instilled (Payer).

7. Camphor and caffeine (Braun, Elsbach, v. Eicken).

8. Ephedrin (B. Martin, Momburg, Grossmann, Domanig).

9. Ephetonin (Heuss, Schloffer, Moerl, Lexer, Schulze, Payr, Plettner, Atanasof, Stahnke) given subcutaneously at the time of avertin instillation.

10. Suprarenin (B. Martin).

11. Venesection (Schmieden, Sebening) followed

(a) by glucose infusion (Schmieden, Sebening, Pels-Leusden, Lexer, Schulze) or

(b) Ringer solution infusion (Schmieden, Sebening, Pels-Leusden).

12. Glucose the day before operation (Denke, Domanig).

13. Cleansing enema (Momburg and Payr).

14. Thyroxin (Pribram, Lexer, Schulze). This according to von Gaza[42] does not interrupt the anesthesia but shortens the after-sleep; Nell[91] and Sebening[92] reported it to be effective only if given for a few days before the operation.

15. Cocaine (2 c.c. of a 1 per cent solution plus 2 c.c. 20 per cent caffeine) is described lately by B. Martin and Kotzoglu,[82] and Seefisch.[121]

16. Coramine. Its action, supposed to be identical with camphor, stimulates respiration and the heart action and is antagonistic to morphine (Uhlmann[135]). Recently Killian,[63] Mörl[85] and Fischmann;[35] Killian and Uhlmann[63a] reported that it was effectual in interrupting avertin narcosis (also: Baum, M. Borchardt, Denk, v. Gaza, Nordmann, H. Schmidt, Unger, Specht).

17. Extract of the thymus (Domanig[25]).

MORTALITY

It seems impossible to elicit the exact number of avertin anesthesias performed and so reliable figures on fatal outcome are unobtainable. Only the manufacturers know how many tribromethanol anesthesias are performed. Their estimate is 250,000. Our estimate from the figures reported is about 40,000 (July, 1931). In this figure, however, the clinics for women are not included (Butzengeiger;[17] Loeser;[78] Stahnke;[126] Beck[7a]). E. Martin[83] performed 4500 anesthesias during labor. The papers about fatal cases (v. Scanzoni;[112] Schrödl;[118] Pribram;[102] Hahn[54]) where death was presumably caused by tribromethanol, have been again and again analyzed and criticized (Nordmann;[95] Glaesmer;[43] Hahn;[54] Kotzoglu;[68] Behrend;[8] Anschütz;[1] Schulze[119]). It is therefore rather difficult to determine what percentage of the deaths have really been caused by the drug.

Anschütz investigated the mortality of 79,750 cases (April, 1932) and came to the conclusion that in only 6 of these was the connection between the avertin anesthesia and the exitus evident.

Indeed, we asked for statements about death cases. We obtained *one* such report (Bsteh[16]) in which undoubtedly avertin was the cause of death. The explanation for this astonishing drop in fatal accidents seems to be better technique and better judgment in the selection of cases.

REPEATED USE OF TRIBROMETHANOL

Repeated use of tribromethanol was reported by Wolf;[144] Lindemann;[76] Knop;[66] Goebel;[45] and Lehrnbecher.[72] Repeated doses of tribromethanol were given in cases of tetanus (Lobenhoffer;[77] Momburg and Rotthaus;[87] Denk;[24] Kaspar;[61] G. Loewe,[79] Schaefer;[113] Uebelhör;[134] Wiedhopf[142]). In cases of subsequent operations on the same individual (Poppert;[101] Lexer;[75] Schulze[119]), in cases of eclampsia (Ruge[110]) and in chorea (Westphal;[141] Enke[33]).

SUMMARY

1. Reports concerning the latest experiences with tribromethanol in the leading clinics of Germany, Austria, Switzerland and other countries abroad are analyzed.

2. Technique in the use of tribromethanol has been standardized, its use is increasing.

3. The rectal administration of tribromethanol is valuable in selected cases; its introduction as a basal anesthetic marks a new era in modern anesthesia.

Table I

CLINICS NOT USING TRIBROMETHANOL

1. Chir. Klinik d. Universität Bern: de Quervain, F.[103]* "Margin of anesthesia too small; mortality too high; uncontrollable; too toxic."
2. Chir. Universitätsklinik Freiburg, i. B.: 300 beds,: v. Brandis, H. I.[14] Killian, H.[14,63]† "Preferring other anesthesias."
3. Chir. Abt. a. St. Krankenhaus r.d.I., Munich, 900 bed hosp.: Grasmann, M.;[49]* "Preferring other anesthesias."
4. Krankenhaus Wieden, Vienna,: Schnitzler, J.,[117]* "Undesirable."

* Head of the Clinic.

† But: see Killian's report on coramine.[63,63a]

Table II

CLINICS USING TRIBROMETHANOL RECTALLY AS A BASAL ANESTHETIC FOLLOWING MANUFACTURERS' INSTRUCTIONS

1. Buergerspital Basel, Chir. Universitätsklinik: Henschen, G.[56]* Christ, A.[19]
2. St. Krankenhaus. Friedrichshain, Berlin: 1201 bed hosp.: Braun W.[15]*
3. St. Hildegard-Krankenhaus, Berlin-Charlottenburg: 138 bed hosp.: Pribram, B. O.[102]*
4. Chir. Abt. a. Krankenhaus Moabit u.d. III. Chir. Universitätsklinik, Berlin: 1025 bed hosp.: Borchardt, M.[13]*
5. Chir. Universitätsklinik der Charité, Berlin, 256 beds: Sauerbruch, F.;[111]* Nissen, R.[94]
6. Lazarus-Kranken-u. Diakonissenhaus, Berlin N., 200 bed hosp.: Seefisch, G.[121]*
7. St. Rudolph-Virchow-Krankenhaus, II. Chir. Abtl; Berlin: 259 bed hosp.: Unger, E.;[137]* May, H.[137]
8. St. Krankenhaus Bielefeld: 453 bed hosp.: Momburg, F.;[86,87]* Rotthaus, E.[87]
9. Chir. Universitätsklinik Breslau: 163 beds: Küttner, H.[70]*
10. Chir. Universitätsklinik Erlangen: 205 beds: Goetze, O.;[47]* Friedrich, H.[30]
11. Diakonissenanstalt Flensburg: 220 bed hosp.: Baum, E. W.[6]*
12. Chir. Klinik d. St. Marienkrankenhaus, Frankfurt a.m.: 310 bed hosp.: Flörcken, H.;[36,37]* Mues, O.[90]
13. Chir. Universitätsklinik im St. Krankenhaus Sachsenhausen, Frankfurt/Main: 323 beds: Schmieden, V.;[116] Sebening, W.[120]
14. Chir. Universitätsklinik Giessen: 239 beds: Poppert, P.;[101]* Glahn, A.[44]
15. Karolus Krankenhaus Goerlitz: 45 beds: Boetesr, O.[11]*
16. Chir. Universitätsklinik Graz: Denk, W.;[24]* Domanig, E.;[25] Uebelhör, R.[134]
17. Chir. Universitätsklinik Greifswald: 120 beds: Pelz-Leusden, F.[97]*
18. Universitätskliniken Halle (Chir. Abtl. u. Nervenklinik): 367 beds: Voelcker, F.;[138]* Gallineck, A.[41]
19. Akademisches Krankenhaus, Chir. Klinik, Heidelberg: 276 beds: Enderlen, E.[31]*
20. Chir. Universitätsklinik Innsbruck: Ranzi, E.[104]*
21. St. Krankenhaus, Chir. Abt., Karlsruhe i.B.: 691 bed hosp.: Haas, W.[53]
22. Chir. Universitätsklinik Kiel: 249 beds: Anschütz, W.;[1,2,3]* Specht, K.;[125] Tiemann, F.[3]
23. Chir. Universitätsklinik Lindenburg, Koeln: 278 beds: Drügg, W.[26]
24. Chir. Universitätsklinik u. Poliklinik Koenigsberg Pr.: 203 beds: Läwen, A.[71]*
25. Chir. Klinik d. Universität Marburg/Lahn: 132 beds: Wiedhopf, O.[142]
26. Staatl. preuss. chirurgische Universitätsklinik Muenster i.W.: 160 beds: Coenen, H.;[22]* Nestmann, F.[93]
27. Jewish Hospital, Riga: Mintz, M.[84]
28. Chir. Universitätsklinik Rostock: 296 beds: v. Gaza, W.[42]*
29. I. Chirurgische Universitätsklinik des Prof. A. Eiselsberg, Vienna: v. Eiselsberg, A.;[29]* Starlinger, F.[127]

* Head of the clinic.

TABLE II (*Continued*)

30. Diakonissenhaus Paulinenstift, Wiesbaden: 233 bed hosp.: Heile, B.[55]*
31. Diakonissenhaus Witten: 330 bed hosp.: Heuss, H.[58]*
32. Chir. Universitätsklinik Würzburg; 980 bed hosp.: Koenig, F.[67]*

* Head of the clinic.

TABLE III

CLINICS USING TRIBROMETHANOL RECTALLY FOLLOWING THEIR OWN METHODS

I. Nordmann's method

1. Chir. Abt. d. Auguste-Viktoria-Krankenhauses, Berlin-Schoeneberg: 760 bed hosp.: Nordmann, O.[95]*
2. Chir. Klinik d. St. Krankenanstalt Bremen: 1450 bed hosp.: Smidt, H.[124]*

II. Kreuter's method

1. Universitaets-Hals-Nasen- u. Ohrenklinik und Poliklinik Charité, Berlin: 100 beds: v. Eicken, C.;[28]* Elsbach, K.[30]
2. Kaiserin Auguste-Viktoria-Krankenhaus, Berlin-Licherfelde: 92 bed. hosp.: Herzberg, M.[57]
3. St. Krankenhaus, Chir. Abtl. Frankfurt/Oder: 350 bed hosp.: Ruge, E.[110]*
4. St. Krankenhaus, Koeln-Muelheim: 263 bed hosp.: Haack, E.[52]
5. Allgemeines Staedtisches Krankenhaus, Nuernberg: 1605 bed hosp.: Kreuter, E.[69]*
6. Deutsche Chir. Universitätsklinik, Prague: Schloffer, H.;[114]* Mörl, F.[85]

TABLE III (*Continued*)

III. Sievers's method

1. Universitäts-Kinderklinik und Poliklinik zu Leipzig, Chir. Abtl.: 350 bed hosp.: Sievers, R.[122]*

IV. B. Martin's method

1. Allgemeines Krankenhaus zu Bamberg: 1300 bed hosp.: Lobenhoffer, W.[77]*
2. Chir. Universitätsklinik Berlin: 223 beds: Martin, B.[81,82]
3. Chir. Abtl. des Stadtkrankenhauses Friedrichsstadt, Dresden A. 24: 613 bed hosp.: Fromme, A.[40]*
4. Universitätsklinik Jena, Chir. Abtl.: 450 beds: Guleke, N.[51]*
5. Chir. Klinik Muenchen: 1819 bed-hosp.: Lexer, E.;[75]* Schulze, W.[119]
6. Kantonsspital Zuerich, Chir. Klinik: Clairmont, P.[21]*

V. Variations of B. Martin's method

1. Rudolph-Virchow-Krankenhaus, Chir. Abtl.: 2591 bed hosp.: Mühsam, R.[89]*
2. Chir. Universitätsklinik Bonn: 162 beds: v. Redwitz, E.[105]*
3. Chir. Universitätsklinik Hamburg-Eppendorf: 2638 bed hosp.: Sudeck, P.;[132]* Roedelius, E.;[109] Schmidt, H.[115]
4. Chir. Abtl. a. Krankenhaus Schwabing, Muenchen: 1910 bed hosp.: Dax, R.[23]*

VI. Payr's method

1. Chir. Klinik der Universität Leipzig, im St. Krankenhaus zu St. Jakob: 465 beds: Payr, E.;[96]* Plettner, F.[100]

* Head of the clinic.

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* Continued from p. 455.

UNUSUALLY LARGE URETER STONES*

A REPORT OF TWO CASES

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THE literature dealing with the diagnosis and the treatment of ordinary stone in the ureter is voluminous. On the other hand, case reports of unusually large stones in the ureter are relatively few, and it is because of their rare occurrence that I wish to present the following two cases.



FIG. 1. Case I. Calculus extending from ureterovesical junction to fourth lumbar vertebra.

CASE I. Dr. S. L. B., male, aged thirty-eight years, entered the Presbyterian Hospital March 16, 1922.

Previous History. Fifteen years previous to admission patient had an attack of right renal colic. An operation was performed but no stone was found.

Thirteen years ago, or two years after the before-mentioned attack of renal colic, a severe pain in the bladder occurred; there was a sudden stoppage of urine, followed by complete retention. A suprapubic cystotomy was done and a bladder stone removed.



FIG. 2. Case II. Enormous calculus in lower end of right ureter.

Present Complaint. Pyuria. The urine has been turbid since the second operation and on examination a large amount of pus was found.

Frequency of Urination. This has been present during the past year, but during the past three months has not been apparent.

Pain in the Bladder. Pain in the region of the bladder radiates to the left thigh and into the kidney. It is periodical and recurs at indefinite intervals. At first relief was obtained by remaining in bed but at present the pain is intensified when the patient remains in bed.

Pain on Urination. Pain is sometimes severe during urination and sometimes continues after urination for a short space of time

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—several seconds. It is severe enough to cause intense discomfort.

Physical Examination. Head and neck, negative. Heart and lungs, negative. Examination of the abdomen reveals a right nephrotomy scar and a midline suprapubic one; otherwise negative. External genitalia, negative. Rectal examination shows the prostate to be small and slightly increased in consistency.

Blood Examination. Leucocytes 14,400; hemoglobin 84 per cent.

Blood Pressure. Systolic 122, diastolic 80.

Wassermann Test negative.

Blood Chemistry. Urea nitrogen 50.0; uric acid 2.2; creatinin 1.5; Non-protein nitrogen 48.0.

Urinalysis. Albumin +, blood +, sugar 0, red blood cells +.

Roentgen-ray Examination. On the left side, a dense shadow occupies the position of a dilated ureter and extends from the fourth lumbar downward into the pelvis (Fig. 1). Three very small shadows are in the pelvis, suggesting calcification of the arteries. Negative for bone change.

Cystoscopic Examination. Right ureteral orifice, normal. Right ureter catheterized without difficulty or obstruction. Left ureteral orifice, elevated; a catheter enters but it is impossible to advance the catheter, due to the presence of a stone in the ureter. Examination of the urine obtained at this examination shows:

	Leucocytes per Cu. Mm.	Cultures
Bladder	6400	Colon bacilli & staphylococci
Right kidney .	20	Sterile

Phthalein Test. Bladder: first one-half hour 36 per cent; second one-half hour 12 per cent; Total 48 per cent.

Right Side: first one-half hour 18 per cent.

Operation, March 29, 1922. The patient was advised to have a nephroureterectomy but consented to a ureterotomy and the removal of the stones. An extraperineal ureterotomy was done and the stones removed.

Result. Recovery uneventful. Discharged from the hospital April 21, 1922.

Case II. D. C. G., male, aged sixty-four years, entered the Presbyterian Hospital, March 4, 1923.

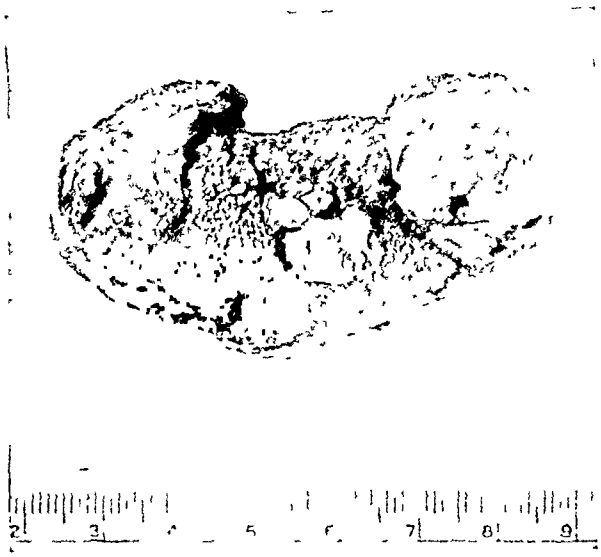


Fig. 3. Case II. Ureter stone after removal.

Previous History. Fifteen years previous to entering the hospital the patient suffered over a period of three years from a severe pain in the left side which radiated down into the penis. These attacks came on about once a month, lasted a day or two following which pus was found in the urine. Roentgen-ray examination at that time was negative for stone.

Present Complaint. Hematuria. Six months before admission the patient noticed blood in the urine, generally decidedly bloody, and at times well mixed with urine. He also passed large clots of blood. The hematuria was intermittent in character, sometimes mild and at other times very severe.

Frequency of Urination. This began at about the same time and has gradually increased in severity. At the present time it is necessary to void as often as ten times during the day and from two to four times at night.

Pain in the Penis. This symptom has been present for six months; it is aggravated by bending forward and also when descending the stairs.

Burning on Urination. This disturbance has been present over a considerable length of time; during the last four or six weeks it has increased in severity, at times being extremely painful.

Difficulty in Starting the Stream. For about six weeks, following catheterization

for the examination for residual urine, difficulty was a daily occurrence.

Physical Examination. Patient is well nourished and appears not to be acutely ill. Head and neck, negative. Eyes react to light and accommodation. Tonsils, atrophic. Liver, palpable 2 cm. below costal arch. Spleen and kidneys not palpable. External genitalia, negative. Rectal examination shows a moderative hypertrophy of both lateral lobes. The margins are smooth and sharply defined. No signs of carcinoma.

Blood examination: Leucocytes 7800; hemoglobin 82 per cent.

Blood chemistry: Urea 30.0; uric acid 3.8; creatinin 1.55; non-protein nitrogen 28.0.

Urinalysis. Albumin +, blood +, sugar 0, leucocytes +++, red blood cells ++.

Roentgen-ray Examination. There is an oval shadow in the region of the bladder, compatible with stone. Another shadow, heavier than bone, suggests an enormous calculus in the lower end of the ureter (Fig. 2).

Cystoscopic Examination. Intravesical hypertrophy of the prostate. Behind the prostate a stone about the size of an unshelled almond. An attempt was made to catheterize the left ureter, but an obstruction was met 1 inch up. Examination of the urine obtained at this examination shows: Bladder: leucocytes per c. mm. 7560; cultures, colon bacilli.

Cystogram. A good filling of the bladder. The solution hides the stone which on earlier films was seen low in the pelvis. A very dense shadow is seen projecting beyond the shadow of the solution.

Phthalein Test. Time in appearance, five minutes; first half hour, 29 per cent; second half hour, 18 per cent; second hour, 10 per cent; total two hours, 57 per cent.

Operation (general anesthesia), March 16, 1923. A suprapubic cystotomy was done. The stone was removed from the bladder and an intra-urethral enucleation of the prostate was carried out. The stone in the ureter was removed by a ureterotomy (Fig. 3).

Result. Recovery uneventful. Discharged from the hospital, April 8, 1923.

Examination of Bladder Stone. A white easily powdered stone. Chemical examination shows this stone to consist of a mixture of calcium phosphate and calcium oxalate, the former predominating, together with a small amount of organic detritus.

Examination of Ureter Stone. A brownish black, rough, irregular stone of hard consistency. Chemical examination shows this stone to consist largely of calcium oxalate with some calcium phosphate mixed with a large amount of organic detritus.

Recurrence. The patient was again admitted to the Presbyterian Hospital, January 28, 1931, eight years after the previous operation. No symptoms were present but he reentered the hospital because he was told by an insurance company physician that his urine contains pus.

Physical Examination. Negative. Rectal examination, negative.

Urinalysis. Albumin 0, blood 0, sugar 0, pus +++.

Roentgen-ray Examination. A shadow in the region of the left ureter. This shadow is an exact duplicate of the shadow found in the left ureter, March 10, 1923.

Remarks. Recurrence of stone. The shadow is identical with the shadow seen before the stone was removed (Fig. 2).

The patient refuses to undergo treatment as he has no symptoms.

COMMENTS

The management of cases of stone in the ureter may be discussed under four headings: (1) Cases in which the stone is small so that in all probability it will pass without cystoscopic manipulation and without surgery. In other words, in a certain number of cases the roentgen-ray examination shows the presence of stone, but from the size of the stone it is reasonable to assume that it will pass without cystoscopic manipulation, especially if it moves after each attack of renal colic, as demonstrated in the *x*-ray film.

(2) In this group may be placed cases in which the stone is of a larger size than in Group 1. In this group we may also discuss cases in which the stone apparently is slow in its descent. Patients belonging to this group may be advised to have cystoscopic manipulation, that is, the use of the ureteral catheter to dislodge the stone, the passing of large catheters or bougies for purposes of dilatation, and the

use of oil injections through the catheter to aid the stone in its descent.

(3) In this group are the somewhat larger stones, hence the choice between cystoscopic manipulation and surgery calls for careful consideration. In dealing with this group excellent results may be obtained by means of the former method, and a stone, moderately large, is often passed where one hardly dares to hope for such a termination.

(4) This group contains cases in which the stone is of such size that the patient is obviously a subject for surgery.

Instances of large ureteral stones have been reported by Kidd, Bugbee, Carsons, Federoff, Fryszman, Stevens and others.

In Collinson's case two calculi with a total weight of 83.2 gm. formed a complete cast of the ureter. Federoff's case showed a stone 10 cm. long, weighing 52 gm., and practically filling the ureter.

The cases reported by Harris and Stevens were unique in that giant calculi were bilateral.

Kidd operated for the removal of a calculus 2½ inches long by 1 inch in width, weighing 1 oz., which had occasioned few symptoms. In discussing this fact the author states that, as a rule, the larger the size the fewer symptoms produced by the stone. Of interest in this regard is Reshower's case where at no time was the left ureter (the site of a giant calculus removed by ureterotomy) obstructed, while the right ureter was obstructed, with resultant hydronephrosis, by a small calculus (freed by cystoscopic manipulation). Harris notes that a number of giant ureteral calculi on record have failed to produce symptoms.

In some cases large ureteral calculi have been successfully removed by ureterotomy. In others, the condition of the kidney and the presence of infection have made ureteronephrectomy necessary.

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PREVENTION OF THE RECURRENCE OF URINARY CALCULI*

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I WISH to present a preliminary report of some work which I have been doing in an effort to avoid that unpleasant surgical sequence where, after the removal of a renal calculus, a recurrence of stone rapidly follows.

I believe we are now ready to agree that in the kidney, as in the bladder, calculus disease is no longer a disease at all, but a symptom. No matter how difficult a surgical problem may grow out of the presence of renal lithiasis, fundamentally, its permanent relief is not to be sought in the simple removal of the stone but demands at the same time an appreciation and correction of the condition which allowed of its formation. So let us look at the factors on which can be placed some reliance as being causative:

First: Urinary stasis takes its place in renal stone formation in equal force and importance as is granted to it in bladder stone. Faulty drainage and urinary stagnation is the essential preparation of the field, possibly present in an unappreciated large proportion of persons, and only awaiting the turn of events for the second link to be forged in the development of the chain of circumstances on which hangs the ultimate development of a calculus growth.

Second: The problem of the chemical character of calculus formation becomes increasingly intricate as one delves into it. No one hypothesis can be stretched to fit all cases. We are practically forced to acknowledge that the chemical change that allows a pure uric acid calculus to form, must differ from that whence an oxylate stone crystallized. As the chemical results vary, so must the chemical cause.

Third: The role played by infection must be separated into its component parts. If it can be proved absent, stasis and chemical changes remain to us. If it be present, it may be expected to vary its influence in accordance with the individual behavior of the specific organism's habits.

Testing these fundamental ideas against our experience with clinical cases has led me to feel that in one variety of stone we are apparently approaching some understanding of its causative factors. This is the so-called earthy, or triple phosphate stone.

Ever since Marcet and Sheele did their early chemical analysis of urinary calculi, we have recognized their observation that once the formation of deposits of triple phosphate takes place, it is very rarely superseded by other salts. Again, this variety is known to be the cause of both the large and the rapidly growing calculi. To these characteristics I believe can be added the observation that once a triple phosphate stone is removed, subsequent stone formation will be of a similar chemical character. I believe these characteristics to depend upon the behavior of certain infecting organisms, and on this basis have evolved a step in preventive therapy, thoroughly tested in allied conditions in the lower urinary tract, checked by laboratory methods for efficiency and safety, and aimed to control and to prevent the infection responsible for this variety of stone.

It is recognized that bacteria often have a very limited range of chemical reaction in which they normally thrive. Change their cultural habitat in this one factor, and bacteriostatic, or bacteriocidal, action is obtained.

* From the Department of Urology, Hospital of the University of Pennsylvania. Read before the Section on Genito-urinary Surgery, N. Y. Academy of Medicine, April 20, 1932.

There is a recognized group of organisms which regularly produce, and then flourish in an alkaline urine, and in this media find a suitable habitat wherein they rapidly change urea into ammonia, with a resulting precipitation of the alkaline inorganic salts of calcium, magnesium and ammonia, of which the characteristic phosphatic calculi are formed.

In order to control this chemical change in the urine favorable for bacterial growth and phosphatic precipitation, we have carried out the following steps in our clinical material in an effort to obtain a prevention of the infection by changing the chemistry of the urine, and in so doing creating a habitat in which bacteria responsible for the same will not grow.

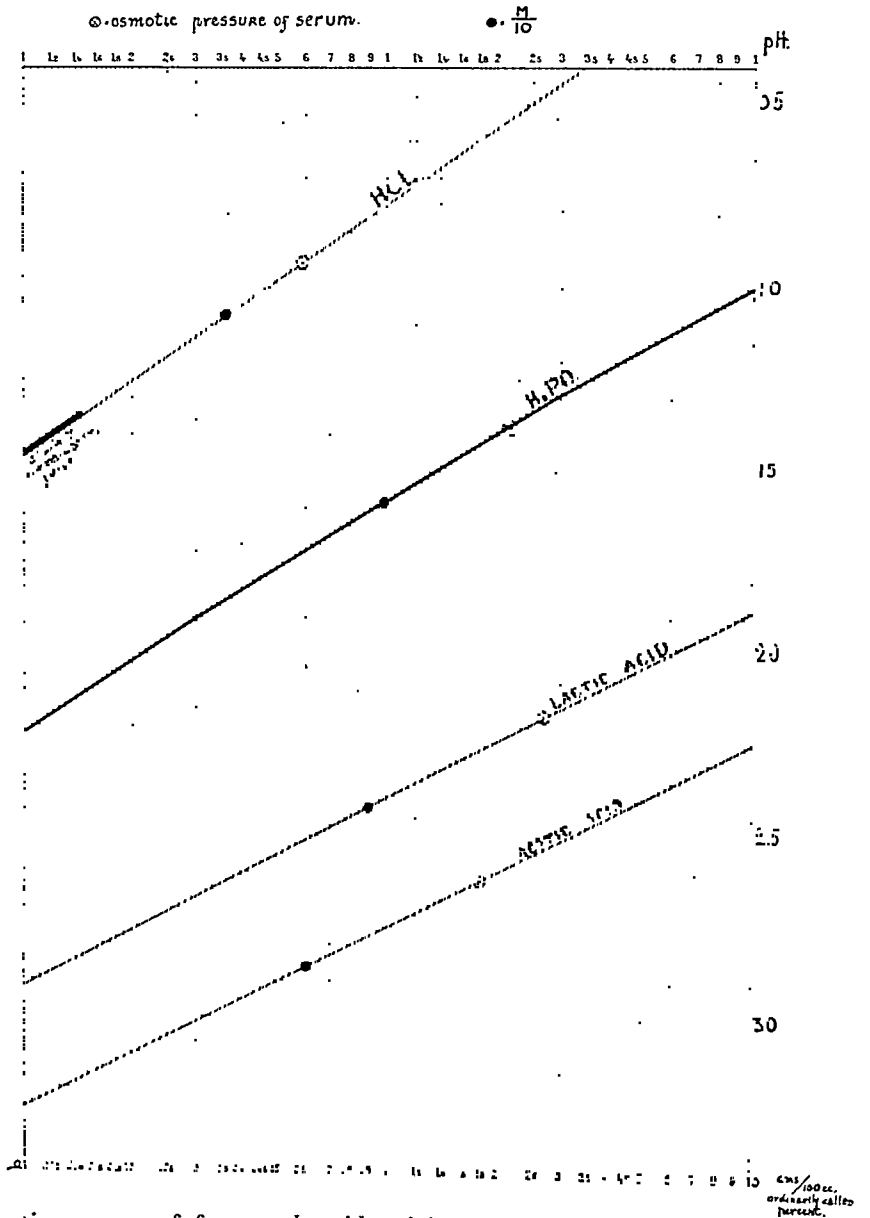


Fig. 1. Comparative curves of four weak acids, giving point of similar osmotic pressure to serum, point of $\frac{M}{10}$, and on right the pH value or acidity, while below is percentage strength or alkali binding capacity.

Our first step was in the treatment of suprapubic fistulae following cystostomy with subsequent drainage. The picture is familiar to you all of the postoperative prostatic patient, whose wound breaks down, creating a surgical menace, that is

a marked detriment to normal healing and closure. These wounds appear to be essentially related to an alkalinization of the urine, and at their worst present an ugly, sloughing gangrenous sore on whose walls and even on the abdominal skin there is likely to be deposited encrustations of phosphates. Some time ago we experienced the ease with which such encrustations could be removed, in fact dissolved, by the topical application of a 5 or 10 per cent phosphoric acid solution. The response in healing following such local treatment is marked; a healthy wound rapidly follows the separation of the ugly slough, with the complete disappearance of phosphatic encrustations. Even in the absence of such breaking-down of the wound, an ammoniacal odor appearing in the dressings, to the initiated, is a warning that trouble is in store. Since adopting this step, the handling of such bladder cases by the topical application of this weak acid solution has made this complication gratifyingly infrequent.

The second step in our clinical handling of these cases was in certain patients where, though the bladder wound was saved from the threatened breaking down, nevertheless, the constant threat of such a possibility was evident in the persistent pool of alkaline urine draining from the bladder. No amount of acidifying drugs by mouth appears to be sufficient in these cases to change the reaction of such an alkaline urine to a normal acidity, while topical applications to the fistula orifice fail of sufficient penetration to reach the bladder urine. It was in such a case, some time ago, that I first attempted a direct irrigation of the bladder cavity with a solution of 1 per cent phosphoric acid. To our surprise it became evident that the bladder not only tolerated this solution without discomfort, but even a stronger solution could be used without marked irritation being experienced by the patient. One or two such irrigations caused a prompt return of normal bladder urinary acidity, and this once obtained it was easily held by the administration of the acidifying drugs by mouth.

This improvement in the handling of these cases has since become a routine in all our suprapubic cystostomies, and I feel it is a definite step in advance in controlling the possibility of postoperative infection with the type of organism which is recognized to have the foregoing characteristics of both alkalinizing the urine and causing a precipitation of the earthy phosphates.

This finding in the lower urinary tract naturally led to our third step in attempting to accomplish the same end in cases of recurrent calculi in the upper urinary tract. As stated before, these stone repeaters practically always form phosphatic stones, and with this in view, I wish to report to you our experiences to date in 11 cases in which we have irrigated the renal pelvis postoperatively with phosphoric acid solution. It is my hope that by so doing we can cause a sterilization of the infection with the organisms which have the afore-described characteristics if they be present, or be a prophylactic against their implantation. Such, I feel, to be a justifiable step towards the prevention of recurrent phosphatic calculus in the upper urinary tract.

In acidifying the urinary passages by irrigation, three qualities are desired in the acid chosen: (1) It should not have to be used in a concentration which would have an osmotic pressure greatly above that of serum for so it would tend to irritate the mucosa by its dehydrating effect. (2) It should have as high an alkali binding capacity as possible to minimize the neutralization by alkali or buffers in the cavity. (3) It should have as high an acidity as the tissues will tolerate.

Phosphoric acid in concentration of 1 gm. per 100 c.c. is approximately 0.100 molar, has a pH about 1.5 to 1.6 and possesses 46 per cent of the osmotic pressure of the serum. This acidity, pH 1.6, is somewhat less than that of normal stomach contents after a test meal which, with a free HCl of 40, have a pH of about 1.4. A solution of hydrochloric acid having the

same pH of 1.6 has a concentration of only 0.019 molar and hence has only 1/5 the alkali binding power of phosphoric acid at this same pH.

Lactic acid at a concentration of 2.7 gm. per 100 c.c. has an osmotic pressure equal to serum but a pH of only 2.2, about one-sixth as acid as phosphoric acid of the same concentration; indeed even at 10 per cent concentration the pH of lactic acid is only 1.9.

Acetic acid is still weaker and when at a concentration of 1.8 gm. per 100 c.c., which has an osmotic pressure equal to serum, its pH is only 2.6; even at 10 per cent concentration its pH is only 2.2. It has about one-thirteenth the acidity of a phosphoric acid solution of the same concentration.

It follows that both acetic and lactic acids are too weak to be suitable for an irrigating solution. On the other hand, a solution of hydrochloric acid will have this pH at so low a concentration that it has only one-fifth the alkali-binding capacity of the corresponding phosphoric acid.

Of these four acids only phosphoric acid fulfills the requirements for an irrigating solution with pH of about 1.6.

Experiments on dogs' kidneys have been performed and repeated by injecting the pelvis through the ureter from a laparotomy incision with a 1, 3 and 5 per cent phosphoric acid solution. One kidney was removed immediately, and the second one removed at the end of forty-eight hours. We have not been able to demonstrate, on microscopic study of these experimental dogs' kidneys, any evidence of damage to the pelvic epithelial lining or the renal papillae; nor is there any evidence in these sections of any caustic action from the use of the drug in the afore-named strengths.

CASE REPORTS

Case 1. S. N., male, white, aged thirty-eight years. In September, 1925, the patient was suddenly taken with an attack of left ureteral colic. A diagnosis of left ureteral stone was made, and a left ureterolithotomy was performed.

Three months later, December, 1925, he experienced a dull pain in the right loin which was subsequently diagnosed as stone in the right kidney. He was again operated upon and seven small stones removed. Subsequent to this he spontaneously passed two small stones.

For the next four years he had mild attacks of right renal pain, lasting from two to six hours. During this interval he was twice admitted to other hospitals for study, but on each occasion he refused operation, and apparently all that was done was a catheter drainage of the kidney, with the subsidence of chill and fever which had accompanied each attack.

His first admission to the University Hospital was on July 17, 1930. Study showed a ureteral calculus in the lower third of the right ureter. After several attempts to aid the stone's passage, a right ureterolithotomy was performed on July 25, 1930, under spinal anesthesia. Convalescence was uneventful and he left the hospital on August 12, 1930. A follow-up cystoscopic examination, on September 8, 1930, showed no obstruction to the right ureter, though the pelvic urine was loaded with pus cells, and a lavage with 1 per cent mercurochrome solution was performed. The patient was re-admitted in November, 1930, remaining in the hospital four days for study. Diagnosis: Right pyelitis, with an *x*-ray shadow suspicious of right renal calculus.

He was again admitted to the hospital in January, 1931, for seven days, complaining of constant right-sided pain radiating down the line of the right ureter. At this time a purulent right pelvic urine was obtained. An *x*-ray plate suggestive of a shadow in the right kidney region was obtained, while low in the ureter appeared an oval shadow similar to that seen in the plates at the time of his right ureterolithotomy. There was no obstruction to catheter passage, nor in the previous, or this admission had there been any fever. His next admission was three months later, in March, 1931, when a more complete study was allowed and a recurrence of his calculus in the lower right ureter substantiated. Two operations were attempted at this time and both failed: the first, from an inability to satisfactorily expose the lower third of the ureter extraperitoneally; and the second, when this was successfully done, the stone was

found to have migrated away from the field of attack.

After further attempts at dilating the lower ureter in the hope that this stone would pass spontaneously, the final operation was performed in October, 1931, and the stone removed from the upper third of the ureter after fixing it by an indwelling catheter. A tube was placed in the ureter up to the pelvis and the wound closed about it. The pelvis was daily irrigated with 1 per cent phosphoric acid and on one occasion a 3 per cent solution was used. There was no evidence of any local disturbance or pain, and when the tube was removed on the eighth postoperative day, primary closure of the wound rapidly followed.

Following his discharge, the patient has reported regularly at intervals of two weeks for right pelvic lavage and ureteral dilatation. During his stay in the hospital, and subsequent to his discharge, he has been put on the regular administration of a dram of cod liver oil twice daily, and urged to continue a diet containing large quantities of milk and eggs. On January 8, 1932, his right pelvic urine was clear of pus, but still weakly alkaline; while on February 23, 1932, it was sterile to culture, and of a pH 6.5. On April 30, 1932, the right pelvic urine was still alkaline to litmus and had a pH of 7.4, while the left pelvic urine was acid to litmus and had a pH of 5.7. This case is still under observation.

Tabulation of Cases

No	Name	Operations	Chemistry	Pelvic Lavage	Drain Removed	Ureteral Lavage	Final Studies
1.	S. N.	1. L. Ureterolithot. 2. R. Nephrolithot. (Voided calculi) 3. R. Ureterolithot. 4. R. Ureterolithot.	Ca. & mg. phos. Ca. carbon.	Daily 1% sol. phosphoric acid Once 3% sol.	8th p.o. day	Every 2 weeks	4 mos. later R. pelvic urine pH 6.1 7 mos. later R. pelvic urine pH 7.4 L. pelvic urine pH 5.9
2.	G. E.	1. R. Pyelolithot.	Uric acid Ca. carbon.	Q.2.d.1% sol.	11th p.o. day	Every 2 weeks	6 mos. later R. pelvic urine pH 6.0
3.	J. W. L.	1. R. Ureterolithot. 2. L. Pyelolithot. 3. L. Pyelolithot. 4. R. Ureterolithot. 5. R. Ureterolithot. R. Pyelolithot. R. Nephrolithot.	Ca. & mag. phos. Ca. oxylate Ca. carbon	Q.2.d.1% sol. Once 2% sol.	21st p.o. day	Every 3 weeks	5 mos. later R. pelvic urine pH 5.8 Has coral calculus in L. pelvis
4.	K. S.	1. L. Pyelolithot.	Ca. carbon. Na. urate	Daily 1% sol.	17th p.o. day	None	2 mos. later L. pelvic urine pH 5.3
5.	S. D.	1. R. Pyelolithot.	Ca. carbon. Triple phos.	Daily 1% sol.	20th p.o. day	None	
6.	F. T.	1. R. Uretero-lithot.		Once 1% sol.	4th p.o. day	Every 2 weeks	
7.	M. G.	1. L. Nephrolithot. 2. R. Nephrolithot. 3. R. Nephrolithot. permanent drain	Triple phosphate	Twice daily for 4 days	Not removed	None	Encrusted nephrotomy sinus. Strong ammoniacal odor. Uremia. Death
8.	H. H.	1. L. Pyelolithot. 2. L. Pyelolithot.	Na. urate Ca. carbon	Daily 1% sol.	17th p.o. day	None	
9.	J. A.	1. R. Pyelolithot.	Ca. carbon.	Q.2.d.1% sol.	6th p.o. day	None	
10.	S. F.	1. R. Pyelolithot.	Ca. carbon. Na. urate Uric acid	Daily 1% sol.	5th p.o. day	None	2 mos. later R. pelvic urine pH 6.0
11.	H. B.	Congenital solitary kidney 1. R. Pyelolithot. R. Nephrolithot.	Ca. oxylate Ca. carbon.	Every other hour for one hour. 1% sol.	23rd p.o. day	None	Fragment in pelvis postoperatively. Attempted to dissolve it to no avail. Removed with forceps on 23rd p.o. day

His recent calculus was composed of calcium and magnesium phosphate and calcium carbonate.

Case II. G. E., male, white, aged thirty-six years, admitted to the University Hospital, November 6, 1931. Diagnosis: Calculus in the right renal pelvis. This calculus occurred in an intrarenal pelvis and at operation difficulty was experienced in getting sufficient exposure for the total removal of the stone, so that during traction the calculus broke and numerous fragments were obtained after careful search. A tube was inserted in the pelvis and the wound closed about the drainage. The pelvis was irrigated daily with a 1 per cent mercurochrome solution, alternating with a 1 per cent phosphoric acid solution. The tube was removed on the eleventh postoperative day. The patient was discharged on November 26, 1931.

Since the date of his discharge the patient has reported at two-week intervals for renal pelvic lavage with phosphoric acid and has been placed upon a diet rich in vitamin A, and cod liver oil in doses of 1 dram twice daily. On February 6, 1932, the catheterized specimen was clear, and of a pH 5.8. On May 1, 1932, the right pelvic urine was acid to litmus and had a pH of 6.0.

The calculus was composed of uric acid and calcium carbonate. A postoperative *x*-ray was negative for any evidence of fragments.

Case III. J. W. L., white, aged forty-two years. The patient was first seen in June, 1919, at which time chronic prostatitis was present, and for which treatment was given at intervals for the subsequent four years. In 1923 dysuria with frequency developed and for the first time the patient reported the passage of four urinary calculi from the right kidney. In previous years, and at this study, his urine was found to be alkaline, with a definite infection in the right kidney pelvis. During the succeeding years, until September, 1928, numerous studies were made in an effort to determine the presence of tuberculosis; as well as an intermittent series of treatments for a persistent chronic prostatitis. During this time the bladder continued to get more and more intolerant, with a persistent alkaline urine and areas of marked ulceration. *X*-rays and pyelograms as early as February, 1923, had shown minute shadows in the lower calyx of each kidney. These were considered to be calculi, but as they measured but 6 × 4 mm. in size on the *x*-ray film, no operative interference was advised. Functional studies repeatedly showed the left kidney to have half the function of the right, and both diminished below normal. Three culture studies of the prostatic secretion remained sterile; while repeated studies for the tubercle bacillus (eleven in all) brought forth but one report of organisms suspected of being the tubercle bacillus. In September, 1928 an acute right ureteral blockage required an immediate ureterolithotomy in the upper third of the right ureter; recovery from which was prompt.

The finding of the one positive culture for the tubercle bacillus, plus his persistent and aggravated cystitis, caused the advice for him to go into the southwest, and while in New Mexico, during the summer of 1929, two operations were performed a month apart for an acute ureteral calculus blockage on the left side.

On his return, in the fall of 1929, *x*-ray examination revealed pocketed calculi in the lower calyx of the right kidney, and two small but suspicious shadows in the region of the left kidney pelvis. On June 4, 1930, a second acute calculus blockage of the right ureter necessitated a second ureterolithotomy to be performed on the right side in the upper third of the ureter. In November, 1930, a uroselectan study demonstrated the growth of the small shadows in both the right and left kidney pelves, but their size was no greater than could be expected to be passed in case dislodgement occurred. In fact, on numerous occasions, small calculi varying in size up to 5 mm. in diameter were spontaneously voided, and during this time the bladder capacity rarely exceeded 4 ounces. On November 28, 1930, a sizable soft stone was passed from the left ureter after a colic of a few hours' duration. During the ten months' interval between November, 1930, and September, 1931, enormous growth of stone took place and a uroselectan study of September, 1931, showed typical coral calculi in both the right and left renal pelves.

On November, 2, 1931, a fall from horseback was sustained and five days later severe colic with complete right ureteral blockage occurred, again associated with alarming clinical symptoms of an infectious character. A catheter was successfully passed by the stone and pelvic drainage obtained, and on November 16, 1931,

a third ureterolithotomy was performed followed by a pyelotomy, with removal of the large pelvic stone; then a nephrotomy into the lower calyx was done with the complete evacuation of the nest of calculi which had been observed there over a period of four years. (*X*-ray studies prior to this operation compared with the studies made two months before showed convincing evidence that the trauma sustained at the time of his fall had fractured the coral stone in his right kidney, a portion of which had caused the right ureteral blockage.)

Drainage was instituted at this operation to both the pyelotomy and the nephrotomy incisions. This drainage was sustained for a period of three weeks and the pelvis was irrigated daily, alternating between potassium permanganate followed by mercurochrome, and a 1 per cent phosphoric acid. It is of especial note that the phosphoric acid caused no renal discomfort, but regularly produced a sense of burning, not unbearable, in the bladder. The drainage tubes were removed at the end of three weeks and the fistula closed four days later. *X*-ray on December 18, 1931, the date of discharge from the hospital, proved the right kidney negative for calculi.

Since his hospital discharge, the patient has reported at ten day intervals for cystoscopic right ureteral catheterization and lavage. It is of importance to record that the first time in the period of eight or more years, the bladder has become tolerant of cystoscopy without sacral or spinal anesthesia. Its capacity has increased from 4 ounces to 14 ounces. The vesical lesions have retrogressed to a point where only an area possibly 1.5 × 2 cm. in size in the fundus of the bladder where the mucous membrane is still redundant, sodden and red. On January 12, 1931, urine collected from the right renal pelvis was brilliantly clear and weakly acid; while the bladder urine was still alkaline. Catheterization on January 28, 1932, showed the right kidney pelvic urine to have a pH of 5.8; while culture showed, on March 28, 1932, from the right side—an organism closely related to encapsulatus pneumonia and B. atrosepticus, and a pH 6.2; and from the left side (still stone bearing) a similar infection, and a pH 7.6.

The last stone removed from the right kidney was composed of calcium and magnesium phosphate, calcium oxylate, and calcium carbonate.

Case IV. C. S., male, aged twenty-nine years, admitted to the hospital February 26, 1932, complaining of attacks of pain in the left loin and left abdomen. These symptoms have been present at intervals over the past six years. *X*-ray showed a calculus in the upper third of the left ureter opposite the lower pole of the kidney. On March 2, 1932, an indwelling catheter was placed to the renal pelvis, following which a left ureterolithotomy was performed under a spinal anesthesia. The stone was found just below the ureteropelvic junction and the ureteral wall markedly thickened. Incision of the ureter failed to find the stone, and incision enlarged to pelvis through which the stone was found in the lower calyx and removed in toto. A No. 16 F. catheter was passed down the ureter and the ureter closed about it bringing it out through the posterior pelvic wall. A larger tube drain was placed in the pelvis through the same pelvic opening. Through the drainage tube the pelvis was irrigated daily with a 1 per cent solution of phosphoric acid. The tube was removed on the seventeenth postoperative day.

Two months later, on April 30, 1932, urine collected from the left renal pelvis was strongly acid to litmus, and gave a pH reading of 5.3.

On analysis the stone was found to consist of calcium carbonate and sodium urate.

Case V. S. D., female, aged fifty-seven years, was transferred from the Medical Service and found to have huge bilateral stag-horn calculi in both renal pelvis. She suffered particularly from pain in the right loin and seemed quite weak and lethargic. An indigocarmine test appeared from the right ureter in fourteen minutes, and from the left ureter in ten minutes, but with only fair concentration of the dye. Phenolsulphonephthalein test gave 20 per cent for the first hour; 10 per cent for the second hour. Blood urea nitrogen 24 mg. per 100 c.c.

Operation was performed on February 24, 1932, and a large stone successfully removed from the right kidney pelvis, by a pyelolithotomy, and a large drainage tube was placed through a nephrotomy incision. She received a daily irrigation of the renal pelvis with 1 per cent solution of phosphoric acid during twenty postoperative days, at the end of which time the tube was removed.

One week after this operation her blood urea nitrogen was 27 mg. per 100 c.c. On March 13, the blood urea nitrogen was 19 mg. per 100 c.c. On March 22, the blood urea nitrogen was 26 mg. per 100 c.c.

Her convalescence was uninterrupted, though she went home, on March 24, against our advice and before complete healing was accomplished. Subsequent observations have not been obtainable.

Analysis of the stone showed it to consist of calcium carbonate and triple phosphates.

Case vi. F. T., male, aged forty-four years, entered the hospital December 3, 1931, complaining of left loin pain. Sixteen years ago the patient suffered a similar attack which was relieved by a hypodermic, and there is no record of a stone being passed. Four months prior to admission he was seized with a severe left abdominal pain which was again relieved by a hypodermic of morphia, and a third attack of similar colic occurred five days before admission, continuing to date of same. *X*-ray study revealed a stone in the lower third of the left ureter, measuring 8 × 6 mm. The catheter obstructed at 8 cm. from the ureteral orifice. His urograms showed no evidence of pelvic back pressure. Nineteen days were consumed in various attempts at cystoscopic reduction and removal. This being unsuccessful, a ureterolithotomy was performed on December 23, 1931, and a solitary stone removed. A catheter drainage was placed to the pelvis through which, on one occasion only, a 1 per cent solution of phosphoric acid was instilled. This drain was removed on the fourth day, and on four occasions, postoperatively, the renal pelvis was irrigated at two-weekly intervals by cystoscopic ureteral catheterization.

The stone was not analyzed, but has the appearance of a calcium oxylate calculus.

Case vii. M. G., female, aged fifty-one years. This patient was admitted to the service of Dr. F. E. Keene on December 13, 1931. She entered the ward with a permanent right nephrostomy sinus. Her previous history recounts an appendectomy, a left nephrectomy for stone, an oophorectomy, a right nephrolithotomy, and later an operation for bladder ulcer and a permanent drainage for a recurrent calculus pyonephrosis of the remaining right kidney, which was performed July 6, 1930. She was in a critical condition from renal insufficiency, and her sinus tract was coated with phosphatic concretions. *X*-ray showed numerous irregular shadows extending down the sinus and involving the remains of a large pyonephrotic kidney. Her urinary drainage was strongly alkaline with a typical ammoniacal odor. After *x*-ray studies and various local measures had been tried, irrigations of the sinus tract with 1 per cent phosphoric acid solution was started on February 28, 1932. These were continued daily for five days without apparent discomfort, and with marked improvement in the wound's condition. The patient died suddenly of cardiac failure on the night of March 3, 1932.

Analysis of the calculus showed it to be of typical triple phosphates. This should have been a test condition in which to have obtained an ideal therapeutic result, and it is regrettable that sudden death defeated the outcome.

Case viii. H. H., male, aged forty-three years, admitted to the hospital March 2, 1932. History recounts a previous left pyelolithotomy for two renal calculi. The patient had suffered soreness and pain in the left side for the past three years. *X*-ray examination revealed two stone shadows in the left pelvis; while urograms showed one to be in the left renal pelvis, and the other in the lower major calyx.

Operation performed on March 9, 1932, demonstrated a stricture at the ureteropelvic junction, through which the incision was carried and two large stones were removed through this pyelotomy opening. A nephrotomy was performed through the lower major calyx and tube drainage brought into the pelvis. Pyelotomy incision was closed about a No. 12 rubber catheter inserted down the ureter and brought out through the nephrotomy wound with plastic reconstruction of the ureteropelvic junction. Daily irrigations with a 1 per cent solution of phosphoric acid were carried on through the drainage tube for seventeen days; the pH of the pelvic urine remaining at 6.0. The tube was removed on the seventeenth day.

Convalescence was uncomplicated. Culture of the urine showed bacillus coli communis.

Analysis of the stone revealed it to be of calcium carbonate and sodium urate.

Case ix. J. A., male, aged twenty-eight years, admitted to the hospital April 4, 1932, complaining of pain in the right side of the abdomen. There had been no previous operations. Study revealed a large calculus in the

right renal pelvis, which was moderately hydronephrotic.

An operation was performed on April 6, 1932, at which time a large stone was removed by a right pyelolithotomy, with a tube drainage to the renal pelvis. Irrigations of a 1 per cent solution of phosphoric acid were carried on through this tube every other day for three applications. The tube was removed on the sixth postoperative day.

Convalescence was uncomplicated.

Analysis of the stone showed it to be of calcium carbonate.

Case x. S. F., male, aged forty-three years, admitted to the hospital March 9, 1932, complaining of pain in the right renal region of six months' duration. Study revealed a stone of elongated shape filling a small intrarenal pelvis and plugging the ureteropelvic outlet.

Operation was performed on March 16, 1932, and the stone removed through a small pyelotomy incision. The only obstruction demonstrated was the adhesion of the ureter to the lower pole of the kidney, with moderate distortion of its course. A small rubber catheter was placed as a drain, through which a daily irrigation of 1 per cent phosphoric acid solution was carried on for the following five postoperative days.

Convalescence was rapid and uneventful. On May 5, 1932 his right pelvic urine was found to be clear with a pH of 6.0.

Analysis of the stone proved it to be of calcium carbonate, sodium urate and uric acid.

Case xi. H. B., male, aged twenty-eight years, admitted to the hospital April 1, 1932, complaining of right loin pain. This patient had been carefully studied before admission and all evidence pointed to the fact that he had a congenital absence of the left kidney, with a large stag-horn calculus in the pelvis and lower calyx of the solitary right kidney.

Operation was performed on April 12, 1932, and after a large pyelotomy incision was obtained, the calculus unfortunately broke on attempted removal. The portion remaining was impacted in the lower calyx. A nephrotomy was then performed over this point and the fragment was successfully removed. Reconstruction of the stone suggested that a third fragment was absent, though thorough search at operation and copious lavage of the pelvis failed to detect it. A large tube drain was placed through the nephrotomy wound to the pelvis and bleeding controlled by a mattress suture on either side of it. No repair of the pyelotomy incision was attempted.

Five days after operation, *x*-ray study revealed a fragment 7 mm. in diameter apparently lying in the pelvis 3 cm. from the end of the drainage tube. Before obtaining an analysis of the stone an attempt was made to dissolve this fragment by intermittent irrigations of 1 per cent solution of phosphoric acid. This was accomplished by passing a ureteral catheter through the lumen of the drainage tube into the renal pelvis. This catheter was connected through a Murphy drip with a transfusion burette, and for the subsequent fourteen days irrigation with 1 per cent solution of phosphoric acid was run through this at intervals of an hour on and an hour off. No discomfort was experienced except occasionally when the patient voided small quantities of urine there was a feeling of slight burning in the bladder and urethra. At the end of two weeks *x*-ray study failed to reveal any change in the stone's size, and a second small shadow was observed in the upper calyx. When comparing this plate with the one taken five days after operation (the latter having been taken in bed without preparation, and was not as clear as could be desired) showed that this second shadow was probably likewise present in the earlier plate too.

Failing of dissolution of the stone, an alligator forceps was passed through the drainage tube and the larger and lower stone successfully removed with the tube at one time. Since we felt somewhat uncertain of the smaller shadow in the upper calyx, the wound was allowed to heal and the patient voided a small stone without colic four days after the removal of the other stone. Analysis of these stones proved them to be of calcium carbonate and calcium oxylate.

SUMMARY

1. Phosphoric acid 1 per cent has a pH acidity of approximately 1.6. Its bacteriocidal value is based thereon.

2. Phosphoric acid 1 per cent is practically isotonic and is slightly less acid than the gastric acidity after a test meal.

3. In experimental dogs, renal pelves injected with 1, 3 and 5 per cent phosphoric

acid fail to show any destruction of pelvic lining epithelium, or damage to the renal papillae.

4. Eleven patients in whom renal pelvic lavage with 1 and 2 per cent phosphoric acid solution has been used, either through a postoperative drainage tube, through a ureteral catheter, or both, have not experienced discomfort or excessive renal pain. The bladder is less tolerant than the ureter or kidney pelvis, and the urethra is the least tolerant of all.

5. The renal pelvic urine in recorded cases has been re-studied and with one exception has been found to retain a normal acidity following this postoperative lavage.

6. This step in prophylactic treatment in cases of recurrent renal calculi has a rationale in both co-related conditions in the lower urinary tract and in bacteriological findings.

CONCLUSIONS

1. By bladder lavage with phosphoric acid postoperatively, alkalinization and phosphatic encrustations can be prevented.

2. The treatment of staphylococcic cystitis, encrusting cystitis, leucoplakia and allied conditions by this means is indicated.

3. The prevention of recurrent renal calculi of the phosphatic variety is being attempted with every indication of success.

4. The possible dissolution of small phosphatic calculi, or fragments left at operation, may be expected by the recognized action of such strengths of phosphoric acid in vitro, and the tolerance of such topical applications in vivo.

5. As a postoperative prophylactic measure against infection with the alkali producing organisms, it is herein proven safe and efficacious in kidney surgery and should therefore aid in the prevention of recurrent renal calculi of the phosphatic variety.

DISCUSSION

Dr. Oswald S. Lowsley: Nothing can possibly be more embarrassing than to have to operate on patients repeatedly for the same condition. It is happening to all of us. The prevention of urinary calculi is one of the most important things that we can consider. It has been our practice for some time to change the reaction of the urine in such patients by feeding them acid or alkaline, depending on the type of calculus found at operation. It is true that in a large stone, when one saws through it, such as a bladder stone, there may be several kinds of material, both alkaline and acid, in the substance of the stone, therefore one cannot in every case be sure that the recurrence of stone will be eliminated by changing the reaction of the urine, but we are quite convinced that in most cases a change in the reaction of the urine will prevent recurrence of stone, because they seem to have a certain specificity. I have mentioned many times a spectacular case in which we had to operate every six months for a period of time. About eight years has gone by now, and this lady, who had had seven operations, five of them by us, has not had a recurrence in eight years, due merely to changing the reaction of her urine. The patient does this herself by taking acid, in her case, and testing her urine with litmus paper. She has learned how to keep her urine acid without going into acidosis, into which we promptly put her when we instituted this treatment.

Dr. Randall has mentioned a most important thing in connection with this, and that is, you must have good drainage. There is no question about it. I remember several years ago Dr. McCarthy mentioned in the discussion of a paper here that his patients were relieved of calcium deposits on wounds by giving phosphoric acid, and so we have been giving it, and it seems to work as well as local irrigation.

I think it is proper in this connection to remember that one must eliminate all possible sources of focal infection. The brilliant work done at the Mayo Clinic in isolating bacteria from the tooth and actually producing stone in the dog's kidneys by injecting these bacteria must make us be alive to the fact that there may be specificity of bacteria, and therefore it is part of our routine always to eliminate every possible source of infection, in the mouth, tonsils, teeth, sinuses, and so on.

Dr. J. A. Killian: For a number of years we have been interested in the problem of urinary calculi from the standpoint of the relationship

of their formation to metabolic reactions in the body, and up to date we have no information of positive value, either concerning their etiology, or methods of preventing their recurrence. However, our work has brought to light a few suggestions which we consider of some practical value. With regard to the change in the reaction of the urine, I think it is important to bear in mind that not only bacteria have the ability to transform urea or amides into ammonia; normal kidney tissue also has that property, and according to the work of Bliss, it is probably most potent of all the tissues in the body in its reaction of de-amidization. Moreover, this ability to de-amidize on the part of the kidney tissue may vary with renal function, either in one direction or the other. At the present time we cannot say whether a diminution of renal function, so far as nitrogen excretion is concerned, is associated with a corresponding impairment of de-amidization. The increase in alkalinity in the urine, due to the ammonia formation, may not be entirely the property of bacteria; it may be due to disturbance in renal function. It is true that vitamin A deficiency changes the character of the epithelium so that it is possible for crystals to become fixed or adherent to the epithelial lining, and the use of cod liver oil to supply the necessary vitamin A is wise, not only on account of its supplying large quantities of vitamin A, but also because it supplies vitamin D, whose principal function is concerned with the fixation of calcium and phosphorus in the body, and of course, preventing the loss of calcium or phosphorus from the body in the excreta. In our advice to urologists to be handed on to patients, what we attempt to do is to avoid changes in the reaction of the urine (we are speaking now entirely of phosphate calculi), changes in the urine in the direction of increased alkalinity, and also to avoid an excess of calcium, magnesium and phosphate which may be excreted in the urine. I think by careful restriction of the diet we can do more to regulate the reaction of the urine than we can by the administration of acids by mouth, with the possible exception of phosphoric acid.

The first thing we do is to avoid the giving of bulky meals, because this is followed by an alkaline tide in the urine. This is greatest following the morning meal, less following the noon or afternoon meal, and it varies directly with the quantity of HCl secreted by the stomach, and the quantity of HCl secreted by the stomach varies again directly with the bulk of the meal. About 1919 Blathewick published a series of observations on the influence of fruits and vegetables on the reaction of the urine, and that list is valuable in the selection of fruits and vegetables which will increase the alkalinity of the urine. In choosing foods as a source of calcium and phosphorus, we bear in mind the fact that all observers who have studied the question of the utilization of calcium and phosphorus by the body, as for instance, Sherman, Mendel, Shohl, etc., have brought out the fact that when calcium and phosphorus are utilized by the body most satisfactorily under the influence of vitamin D, they are utilized or stored in about the same proportion in which they occur in milk; usually two parts of calcium to one part of phosphorus. In regulating our calcium and phosphorus in the diet, we attempt to observe that ratio.

Another important thing is this: it has been recently shown that probably the phosphate which occurs in the urine is not all filtered from the blood. It may be formed by the action of kidney tissue on hydrolyzable phosphates in the blood. We therefore avoid giving a large quantity of carbohydrate with any one meal because with a large supply of carbohydrate we have an increase in the hydrolyzable phosphates in the blood which furnishes available material for an increase of phosphate in the urine.

Dr. Joseph F. McCarthy: Dr. Killian and I have conjointly made it our rule to have the most precise quantitative and qualitative estimations made of every stone removed; Dr. Killian then attempts to regulate the patient's metabolism as nearly as possible from the biochemical standpoint. For quite a number of years we have been using acidulated fluids in the bladder cases mentioned by Dr. Randall with considerable success. I feel we are much indebted to Dr. Randall for carrying out that idea to its logical conclusion, the renal pelvis. Personally, I think there are three factors: (1) the factor of obstruction, the primary factor; (2) the biochemical, and (3) the factor of infection.

Dr. Victor Cox Pedersen: The element of infection has been spoken of throughout the evening as to teeth and tonsils, but not materially as to the intestines. I think we do

not do our duty by any of these cases unless we investigate what is going on in the intestinal canal. Over and over again we find a very active disorder, usually in the form of the failure of balance of the bacteria that belong there, the B. coli group making up from 65 to 95 per cent, associated with some of the ordinary pus-producing organisms. Here we have a case in point. A man sixty-three years old, with no teeth, tonsils out, suddenly develops hematuria. The phenolsulphonephthalein test is normal; the urine is normal except for blood and numerous B. coli, staphylococci and enterococci. The intestinal tract shows very active fermentation of carbohydrate with butyric acid fermentation associated with it, and some putrefaction. All that is necessary in that man is obstruction somewhere in his urine-secretory apparatus to produce pus there, either as a general infection of the mucous membrane or a single focus of pus in the kidney, or multiple foci, or the precipitation of salts in the formation of stone. By paying attention to his diet and using urinary antiseptics we have so far brought that man far along in the line of convalescence. If we can evolve for him the right kind of diet, that man should never again have any trouble from his urinary system, but it was a hotbed of infection. I believe we are erring when we do not go into the intestine for one of the sources of trouble in all cases of kidney trouble.

Dr. Randall (*Closing*): The subject of stone is certainly a baffling one, and no theory is going to solve the question in its entirety; the causes are too diversified. My remarks have been limited to phosphatic stone. I do not think that dietary regime as a treatment or prevention is going to do anything of great benefit. It has been tried so many times and in so many different ways. But that it is a factor in stone formation cannot be denied. I have been trying to read into stones the picture which most of us have of the necessity of finding an obstruction, and two weeks ago, in removing a kidney stone and being unable to demonstrate the reason for its being there, I had the opportunity of apologizing for my theory before the class. I expounded on the idea of the dietary question as an answer in this patient. The chap was under spinal anesthesia, and I was talking not to him, but to the class, of the likelihood of dietary reasons being in his case the cause of stone formation, and the patient spoke up and said: "You certainly hit it on the head." He ran a restaurant, and ate at the most irregular hours and irregular food, and he said: "Moreover, my sister has had a stone removed from her kidney, and my father died of a kidney stone." His was not a phosphatic type of stone, but that there are factors, such as diet, which enter into the problem, must not be denied.

This study has three reasons for its presentation: First, it begins to show that the renal pelvis has been a *noli me tangere* for too long a time, and I believe we will learn to do more in the way of local handling of pelvic troubles in the future. Secondly, I feel that the use of phosphoric acid in the way outlined is a safe step in preventive treatment, and a prophylactic against secondary phosphatic stone formation. Thirdly, it may be curative in those cases where alkaline forming organisms already dominate the field and are causing recurrent precipitation of phosphate concretions.

PRESENT STATUS OF SURGICAL TREATMENT IN PULMONARY TUBERCULOSIS*

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IN a paper published in 1822, Sir James Carson, this early pioneer in the treatment of tuberculosis, said; "It has long been my opinion that if ever this disease is to be cured, and it is an event of which I am by no means disposed to despair, it must be accomplished by mechanical means."

This prophecy has been, partially at least, fulfilled. We at present possess efficient surgical means by which mechanical collapse of the diseased lung and cure of tuberculosis can be accomplished. Pneumothorax, thoracoplasty, apicolysis, partial thoracoplastic collapse, multiple intercostal neurectomy, pneumonostomy and possibly pneumonectomy are the means by which remarkable results have been so far obtained.

If I might use a metaphor I would say that the possession of arms and ammunition is not sufficient for the winning of a war. The art of using them efficiently is of greater importance than their mere possession. The science of ballistics is far more important than the discovery of explosives and without an exact knowledge of their proper use our weapons may be more of a menace than an aid.

When dealing with the surgical treatment of pulmonary tuberculosis, and perhaps with thoracic surgery as a whole we surgeons must understand that the pioneer period is past. We enter the ballistic period of this method, if I may use this expression, which is the thorough knowledge of physiology and pathologic physiology of the two principal functions we have to deal with, namely, respiration and circulation and more especially, as thorough a knowledge as possible of the pathology and clinical evolution of pulmonary tuberculosis. The heroic period of rib-cutting and surgical gymnastics has likewise passed. Thoracic surgery today is a highly specialized branch in which the surgeon must possess a great deal of theoretical and special technical knowledge, and furthermore get the habit of working hand in hand with the medical man, the bronchoscopist, the roentgenologist and the pathologist; each individual problem should be discussed in common, so that the indication for operation can be reached and even the kind of operation determined. The surgeon is no longer permitted to say to the medical man, "If you cannot do anything for that patient, let me try surgery upon him." Moreover it is my belief that if we wish to have our patients benefited by our progress in thoracic surgery we should first gain their confidence and more especially the confidence of our colleagues in medicine. The surgeons operating in large tuberculosis hospitals know well how difficult it is to convince the patients to be operated upon and the poor moral effect upon them of an unsuccessful attempt; on the contrary it becomes easy to sell them the idea of operation after a series of operative successes. On the other hand the medical men and the phthisiologists can afford no longer to ignore the resources of modern thoracic surgery, even in apparently desperate cases which seem beyond surgical help. Therefore the cooperation of those practicing thoracic surgery tempered with a degree of boldness, and of broadminded and progressive phthisiologists is the ideal

* From the Thoracic Surgery Departments of Metropolitan and Seaview Hospitals, N. Y. Read at the Convention of the N. V. A. Sanitorium, Saranac Lake, N. Y.

scheme for promoting and developing surgical therapy in tuberculosis. If we wish to advance the efficiency of surgical treatment of tuberculosis by extending its indications and increasing its effectiveness, phthisiologists must become more surgical minded and thoracic surgeons must learn more and more about the physiology and pathology of the chest organs.

With these ideas in mind I am not going to deal with surgical technicalities, in so far as I can avoid them. Nor do I intend saturating this paper with literature with which the reader is familiar. I shall try to present the results of the experience of our medicosurgical chest group, obtained at an active center of about 2000 beds comprising the tuberculous services of Metropolitan and Seaview Hospitals, in which 8 to 15 major operations are performed every week, and in which several hundred cases with pneumothorax are constantly and carefully followed. My topic will be more especially the scope and indications of modern surgery in pulmonary tuberculosis.

IMPORTANCE OF CLASSIFICATION

It is evident that discussion of this subject cannot be placed upon solid foundations unless we first agree upon a comprehensive classification of tuberculosis, both pathological and clinical. Talking about surgical treatment of tuberculosis is, today, something as vague as discussing the surgical treatment of tumors in general. The differences between benign and malignant tumors are by no means greater than the differences between various forms of pulmonary tuberculosis.

I agree with Tendeloo that a clean-cut classification of pulmonary tuberculosis is impossible. However, having had the privilege of working for the past year in close cooperation with Dr. George G. Ornstein and his staff I am strongly impressed by the usefulness of the classification elaborated by him and Drs. Ulmar and Dittler. Their division of pulmonary tuberculosis into four forms—caseous pneumonic, exudative, exudative productive and chronic productive, is in my experience a proven clinical reality; it offers the great advantage of a solid foundation upon which our operative indications can be determined, and upon which our results, successes or failures can be better understood.

The caseous pneumonic form of tuberculosis corresponds to the ulcerocaseous or fibrotic forms of different authors. Caseation, expulsion of the sloughed-out material and formation of cavities are its characteristics. Fever and toxic symptoms generally subside with the elimination of sloughed-out pulmonary tissue, and the general condition of the patient improves. But the cavities remain open, sputum is persistently positive and spontaneous cure under medical treatment is exceptional, if possible. This is what I might call the one and only surgical form of tuberculosis in which surgical treatment is indicated.

The exudative form, which is an acute type of temporary duration, represents an intense allergic reaction in a hypersensitized lung due to a small superimposed tuberculosis infection of a patient hypersensitive to the tubercle bacillus or its products. Massive infiltrations are often present; its beginning might be clinically and radiographically identical with the onset of the caseous pneumonic form. Annular shadows are frequently present in the radiograms, and are often impossible to differentiate from real cavities, although the scarcity of physical signs is in favor of the former. Only the evolution of the disease can solve this diagnostic problem which is of paramount importance for the choice of treatment. In fact, a few months or oftentimes a few weeks of complete rest will suffice to clear up these signs and cause the annular shadows to disappear. The fundamental differences between caseous pneumonic and exudative forms is that in the latter there is no necrosis and sloughing out of pulmonary tissue but only an infiltration which heals by resolution. Surgery has not and must not have anything to do with this form. There is no doubt in my

mind that the bulk of "successes" after phrenicectomies refer to this form and that the "miraculous cures," as we shall see later, have been obtained not because of, but in spite of, the operation. The same holds for pneumothorax or any other form of collapse therapy which cannot logically be indicated for a consolidated and non-collapsable lung.

The third form is the *exudative productive* in which after absorption and healing of the inflammatory process, which may be very slow, strands of scar tissue are left behind. But there is no tendency to softening or cavity formation and consequently no indication for collapse therapy.

The fourth form is the *chronic proliferate or chronic productive*, corresponding to the acinonodosal form of Aschoff. It is the result of small superinfecting doses in a relatively non-allergic tissue, essentially chronic, accompanied by very few symptoms and is little influenced by any form of treatment, except in some cases in which small cavities are present.

It is obvious that for these last two forms there is no indication for surgical treatment, except when caseous pneumonic tuberculosis is associated with them; consequently surgical cures reported as having been obtained in these two forms of tuberculosis cannot be given serious consideration because surgery cannot influence their course.

The foregoing considerations justify the contention that only a comprehensive classification will enable us to understand one another when we discuss indications and results of surgical treatment of pulmonary tuberculosis and that reports of operative cases in which the form of tuberculosis is not clearly indicated cannot be considered of any scientific value.

Surgical treatment of tuberculosis should thus be limited exclusively to caseous pneumonic forms and more especially to what Ornstein, Ulmar and Dittler designate as the "end results" of this form, that is the honeycombed lung or that with cavitations the size and localization of which may vary. Indications and methods vary according to the degree and location of the lesions, the resistance of the patient and the presence or not of pleural complications. Serous fluid or even purulent tuberculous fluid developed spontaneously or following artificial pneumothorax does not greatly influence operative indications; mixed infection, on the contrary, and especially when septic or anaerobic infection with or without internal or external fistulae is present, does influence the treatment. Because of their importance the surgical treatment of these pleural complications will be touched upon separately at the end of this paper.

Surgical treatment in caseous pneumonic tuberculosis aims at the anatomical collapse of pulmonary cavities and is successful only when this result is obtained. The methods of collapse therapy are: artificial pneumothorax, phrenicectomy, intrapleural or extrapleural pneumolysis with or without "plumb" and thoracoplasty, paravertebral partial, total or enlarged. Other methods, more radical aiming at the suppression of the diseased area and more in the domain of the future than of the present, are pneumonostomy and pneumonectomy, that is opening and draining of intractable cavities or surgical removal of the diseased portion of the tuberculous lung.

PNEUMOTHORAX

Pneumothorax produces collapse of the lung by suppressing the normally negative intrapleural pressure. The lung, a perfectly elastic organ, collapses when air is introduced in the pleural cavity by the same mechanism as when an opening is made in the thoracic wall. It does not collapse because it is "compressed" by the air introduced into the pleural cavity but because it is no longer forced to expand by the negative intrapleural pressure. In fact, the lung always tends to collapse and will do so unless it is forced to do otherwise. So that when we talk of "compression" of the lung in artificial pneumothorax we

merely forget that within an hour or so, after positive intrapleural pressure is artificially produced, the intrapleural pressure again becomes negative because of the active interchange of gasses through the pleura. Only in the cases with very thickened pleura can positive pressure be maintained for a longer time. It is not the same, of course, with spontaneous pneumothorax in which tension pneumothorax is often present due to the one-way valve mechanism of the pleuropulmonary communications. In these cases air accumulates in the pleural cavity and its pressure can become equal to the pressure developed in cough, that is up to +60 or +80 mm. Hg, which is a tremendous positive pressure when compared to the −6 mm. Hg of mean normal pressure. However even in these cases, when the internal fistula is sealed up even for a short while, the pressure very rapidly falls, parallel with the fall of partial pressure of O_2 which from 20 per cent drops to 5, 4, 3, 2, 1 per cent, and even less. Studies of the O_2 and CO_2 percentage in the gases in pneumothorax carried on at Metropolitan Hospital with the cooperation of Drs. Kontrowitz and Levine, and at the Surgical Research College with Dr. Birnbaum, have convinced me that prolonged "active compression of the lung" by artificial pneumothorax is an *ignus fatum*, a physiological impossibility. The logical conclusion of these facts is that frequent refillings, even with moderate pressures, are far more efficient in collapsing the cavities and maintaining the collapse than sporadic ones even with high positive pressures.

With these restrictions, I admit that artificial pneumothorax is the most efficient means at our disposal for collapsing pulmonary cavitations; this however depends upon other factors which may prevent collapse. Adhesions of the lung to the thoracic wall (or more exactly of the visceral to the parietal pleura), or very thickened visceral pleura, or central location of the cavities may render impossible a satisfactory selective collapse. In these cases, if after a month or two, good selective collapse is not obtained it is useless and even dangerous to continue the refillings: it is useless because the absence of effective collaps does not favor cure; dangerous because if the sputum remains positive a spread of the disease to the other lung or even to the already collapsed lung is always a possibility. Furthermore, spontaneous pneumothorax can occur at any time in the course of an artificial pneumothorax, which is always a serious and often fatal complication even in patients so far apparently in good condition. The use of *oleothorax* with or without gomenol oil for the purpose of thickening the pleura and preventing spontaneous pneumothorax has not, thus far, given us satisfactory results. Therefore I believe that it is useless and dangerous routinely to prolong pneumothorax treatment under these conditions and lose that valuable period during which other and more efficient methods can still be effectively used. It is time I, believe, to react against indiscriminate use of this otherwise most useful therapeutic procedure.

Our group of workers believe that pneumothorax should be avoided in the very early periods of acute tuberculosis for the following reasons: (1) because early diagnosis between exudative and caseous pneumonic forms is often very difficult; (2) because even with the caseous pneumonic type in its early stages it is impossible to compress a lung which is still consolidated and in which cavities as such are not yet present; (3) it is especially in these early forms that spontaneous pneumothorax most frequently complicates artificial pneumothorax.

Furthermore we believe that in chronic cases of caseous pneumonic tuberculosis indications of pneumothorax should take into consideration the degree of destruction of the parenchyma of the affected lung. We come more and more to the conclusion that when the degree of destruction of the lung parenchyma is such that there can be no reasonable hope for effective re-expansion, an early thoracoplasty may be

the treatment of choice. In fact, in such cases, where pneumothorax has been maintained for several years, the healed lung is often so thoroughly organized that when the air is allowed to be absorbed, mediastinum, heart and trachea are displaced to the affected side and dyspnea due to the traction upon the heart and to vicarious emphysema of the other lung develops. In these cases we are obliged either to maintain the pneumothorax indefinitely or to perform a thoracoplasty. The question we are studying at present is whether in these cases a definite thoracoplastic collapse, which will save the patient often several years of inactivity and protect him from complications which are always possible, is not the best solution of the medical and economical problems involved.

To summarize, we can state that artificial pneumothorax is indicated only in caseous pneumonic forms and only when effective collapse can be obtained. Otherwise pneumothorax should be completed or replaced by other surgical measures.

PHRENICECTOMY

Paralysis of the diaphragm by surgical procedures upon the phrenic nerve is produced with certainty only by avulsion of the nerve. Block by alcohol injection, by crushing or simply by sectioning the phrenic nerve, aimed at the production of temporary paralysis of the diaphragm is inefficient in at least 50 per cent of the cases. The presence of one or more accessory phrenic nerves, as well shown by Felix, is not always affected by these procedures unless very skillfully performed, so that paralysis of the diaphragm does not occur and the operation is useless.

Phrenicectomy produces immobilization of the diaphragm in an elevated position, loss of its muscular tonus and muscular atrophy. The degree of elevation depends, naturally, upon the difference between intrathoracic and intra-abdominal pressures. When the lung is healthy, as when phrenicectomy is performed for intractable hiccough, very slight elevation of the diaphragm occurs. On the contrary in tuberculosis in which the lung shows fibrotic retraction, the diaphragm will rise considerably, being aspirated into the thoracic cavity because of the lowering of intrathoracic pressure due to the shrinkage of the lung; this occurs by the same mechanism as in massive atelectasis, and, as in this condition, not only the diaphragm but also the trachea, the heart and even the thoracic wall are displaced towards the affected side. The proof of this is given by the well-known fact that oftentimes in cases of fibrotic or atelectatic shrinkage of the lung we find the diaphragm, without phrenicectomy, more elevated than it is in exudative or productive forms after phrenicectomy. For these reasons it is an open problem whether fibrosis of the tuberculous lung and cure of the disease is the result or the cause of the rising of the diaphragm. At any rate as it is inadmissible that an inert membrane such as the denervated diaphragm can play any active role in its rise we must look elsewhere to justify the possible usefulness of this operation. The proponents of phrenicectomy contend that collapse of the cavities and cure are produced by compression, relaxation and immobilization of the lung, and by modifications in the physiologic action of cough.

Let us see how effective these factors are.

There is no doubt that phrenicectomy produces but a slight decrease in the capacity of the chest, equal to a pneumothorax not exceeding 200 to 300 c.c. of air. It is obvious that this cannot produce by any means an effective degree of pulmonary collapse. As for the so-called "relaxation" I confess that I cannot understand its mechanism. Slight decrease in the size of the lung and slight decrease of its elastic recoil cannot produce collapse of large cavities in the midst of a portion of lung parenchyma which is more resistant than the healthy parts of this organ. We know that often we cannot obtain an effective collapse of cavities even with successful pneumothorax in which 1000 to 1500 c.c.

of air are maintained in the chest, or with thoracoplasty, when they are central and covered by a considerable amount of healthy tissue. How then can we admit that a paralysis of the diaphragm, equivalent to a small pneumothorax, can bring about the collapse of an apical or even a lower lobe cavity? The only method by which closure of a cavity could be produced would be kinking of the "drainage bronchus of the cavity" and absorption of the air contained therein, as Alexander, Laurell and myself have shown. Phrenicectomy has never been able, clinically or experimentally, to produce a degree of collapse of the healthy lung sufficient to cause atelectasis even in the portions of the lung which are in direct contact with the diaphragm, as 400 or 500 c.c. of fluid in the chest will do. For these reasons "relaxation," if possible, is of negligible importance. It is rather one of those more or less metaphysical expressions which unfortunately only too often have found their way into pulmonary pathologic physiology and which show how little clinicians often care about physiology.

Similarly immobilization of the lung produced by phrenicectomy is much less important than is generally believed. There are at present in the literature a number of cases in which double phrenicectomies had been performed at one sitting for hiccough. Experimental cases of bilateral phrenicectomy are legion. In neither of these instances have asphyctic phenomena been noticed. This shows that the intercostal and accessory respiratory muscles can easily replace the paralyzed diaphragm. Furthermore Duchenne de Boulogne showed, over fifty years ago, that very rapidly after phrenicectomy the activity of the lower intercostal muscle increased to such a degree that even the inspiratory expansion of the costal borders which is due to the action of the diaphragm and which has temporarily decreased after phrenicectomy returns to normal after a short time. This transient and relative immobilization of the lung may explain to some extent the temporary improvement often noticed after phrenicectomies; so that the most we can say in that respect, is that phrenicectomy cannot produce immobilization of the lung unless by other procedures the intercostal muscles have already been paralyzed as after multiple neurectomy or thoracoplasty; phrenicectomy *alone* cannot permanently immobilize the lung.

In the same way, the modifications of cough action due to phrenicectomy are so variable in character and degree as to have no definite importance. We have seen in our cases and in those reported in the literature that some patients expectorate more, some less, some easier than before, some with greater difficulty and in some no change has been noticed. From the foregoing considerations it appears that the therapeutic influence of phrenicectomy upon the course of caseous pneumonic tuberculosis, the only type in which it is indicated, is almost if not entirely nil. In 150 cases of caseous pneumonic tuberculosis operated at our center by different surgeons, and examined from six months to five years after operation by Dr. Ulmar and myself, we have not been able to discover a single case of permanent cure due to phrenicectomy. Therefore I believe that the so-called numerous "cures" reported by tuberculosis centers in which this operation is performed in an indiscriminate way and as a routine operation are hasty reports dealing with cases which showed slight and only temporary improvement; or they concern cases of exudative forms of tuberculosis in which cure has occurred by itself as in cases I showed at the beginning. Does this mean that we should exclude this operation from our surgical treatment? To this question I answer, yes, if we count on it alone to cure our patients; precious time is lost in waiting for results, exactly as with an inefficient pneumothorax; in both instances, other and more effective procedures should be added, and this before the proper time to act is past. On the contrary, phrenicectomy when used in conjunction with other operations such as thoracoplasty is useful provided that we do not expect from it more

than it can give. Some will probably object that a distinction should be made between apical and basal lesions, and between a thin and thick walled cavity. I do not believe this, and at least our results do not permit us to accept this distinction. Among the cases I referred to, there is an imposing number of thin-walled apical and basal lesions, in which no definite collapse of the cavities was produced and no permanent cure was obtained.

The results of phrenic exeresis in hemoptysis are equally problematic. Hemoptysis is so capricious in its onset, duration and severity, and often stops so suddenly, that it is difficult to formulate a definite statement upon the efficiency of phrenicectomy in it.

PNEUMOLYSIS, INTRAPLEURAL OR EXTRAPLEURAL

INTRAPLEURAL. In cases of pneumothorax, in which collapse is prevented by adhesions, Jacobaeus, Unferricht, Kremer, Matison, Maurer and others, have devised and perfected a method of intrapleural section of the adhesions, guided by a special endoscopic instrument, similar to the cystoscope, known as the thoracoscope. Sections of the adhesions can be done with the electric cautery or by high-frequency coagulating and cutting electrodes. There is no doubt that in well-selected cases where the adhesions are thin, avascular and sufficiently long so as not to expose the patient to the danger of hemorrhage or section through the diseased lung parenchyma and consequent production of pleuropulmonary fistulae with spontaneous pneumothorax and empyema, this method is useful. In 12 of our cases a perfect collapse was obtained after section of adhesions, and were followed by clinical improvement with negative sputum, in spite of the development of aseptic fluid in the chest in one of them. I therefore subscribe to what Sauerbruch has said, namely, that this procedure, in carefully selected cases and when skillfully performed, may give valuable results.

Another method of intrapleural pneumolysis consists in the section and ligature of the adhesions after thoracotomy. The consensus of opinion is that this is often a dangerous procedure in tuberculous patients. Thoracotomy for tumor of the lung is harmless, because in these cases the pleura is healthy; on the contrary in cases with tuberculous pleura often after thoracotomy fistulae are formed leading to tuberculous empyemas with superimposed infection by pyogenic organisms.

EXTRAPLEURAL: More efficient and, I believe, more promising are the procedures designated under the name of extrapleural pneumolyses, especially when applied to apical lesions where pneumothorax has not been able to collapse the adherent apex.

The objective of extrapleural pneumolysis, or more exactly of apicolysis, is the selective extrapleural collapse of the diseased apical lesion alone, whereas the healthy part of the lung is left untouched. Three methods thus far have been proposed for surgical collapse of apical cavities.

1. *Extrapleural Apicolysis.* This procedure consists in stripping the parietal pleura of the apex from the chest wall and filling the space with fat, homogenous or autogenous, or with a pendunculated muscular flap or better with the paste of Baer called by the German authors "plombe"; this is a mixture of paraffin fusible at 48°C., with 1.5 per cent of neutral bismuth carbonate and 1.5 per cent vioform. This operation is performed under local anesthesia by posterior paravertebral incision or anterior under the clavicle. If the intercostal space is not wide enough to permit the introduction of a finger, a 2 inch length of the second rib is resected. Posteriorly the third rib should always be resected.

2. *Scalenectomy.* The second method is scalenectomy with resection of the first two or three ribs, by anterior approach. It was devised by Jay Coffey in this country and Lawers in France. The incision is made above the clavicle; after

section of the clavicular portion of the sternomastoid muscle, both scalene muscles, anterior and posterior, are sectioned and the first rib is resected for its entire length. Thus the apical pleural dome is definitely deprived of its suspensory systems, so that after being stripped from the chest wall it has no tendency to re-expand. In this procedure, which is carried out under local anesthesia there is no necessity of any filling or paste; furthermore there is no danger either of tearing the pleura or of injuring any of the important vessels or nerves which lie on the apical dome, as is possible in the previous method.

3. The third procedure, advocated by Maurer, Rolland and Proust in France, consists of a *complete resection or even disarticulation of the first two or three ribs* by a paravertebral approach. It is therefore more of a partial upper thoracoplasty than an apicolysis.

I have performed 16 apicolyses by the first method without any postoperative mortality or morbidity. In 3 the cavity left after stripping of the pleura was filled with pedunculated muscular flaps and gauze packing; in the remaining cases Baer's "plombe" was used. The oldest patient was sixty years of age, the youngest was sixteen. All have markedly improved and 30 per cent are now bacilli free. Three patients had bilateral lesions. In one a girl eighteen years of age apicolysis was performed on the right side, and a week later pneumothorax was induced on the left side followed by a good collapse of this lung, and marked improvement of the general condition. In one a bilateral plombe has been done, and in the third the other side will be performed later.

The possible complications of this method are: (1) suppuration due to the paste and its elimination; (2) perforation of the roof of the pulmonary cavity and penetration of the paste down in to it; (3) forcing of adhesions by the weight of the paste and migration of it lower in the chest. The second and the third complications are exceptional, and they expose the patient to a serious danger. I never have had any of these accidents with this method. On the contrary, slow expulsion of the paste from the chest, so that it gradually bulges under the skin, forming a tumor the size of which varies from that of a walnut to a hen's egg frequently occurred in my cases, necessitating the extraction of the protruding paste under local anesthesia. Serious suppuration never occurred, although in 3 cases I had fistulae which had persisted during several months. I think that this method constitutes a real progress in surgical treatment of tuberculosis because of its benign nature, its efficiency in one-third of the cases and especially because of its possibilities in bilateral cases. Its principal indications are: (1) cavities or lesions of the apex, with the rest of the lung free, when these lesions are located above the third rib anteriorly and when previous pneumothorax did not collapse them; (2) in bilateral apical lesions; (3) in apical lesions in one lung with more advanced lesions in the contralateral lung, but which can be collapsed by pneumothorax or even by thoracoplasty. In these cases, apicolysis is certainly a better method than bilateral pneumothorax, or phrenicectomy on the side of the apical lesion and pneumothorax in the other. The fourth, which according to Sauerbruch, Felix and Niessen is its principal indication, is in cases in which thoracoplasty has not produced a complete collapse of the apical lesions. Conversely, if apicolysis has not completely collapsed the apical lesions there is no contraindication whatever in later performing a thoracoplasty. I do not believe that apicolysis is a procedure as efficient as an upper thoracoplasty, but it has the advantage over this operation of being far less serious.

Multiple Intercostal Neurectomy

This operation, devised by Alexander, consists in the resection of the intercostal nerves of the diseased side. It is a technically easy operation, and generally harmless. Combined with phrenicectomy it assures a good immobilization of the chest. It is

unable however to produce any kind of adequate collapse, therefore its field is limited to the cases in which nothing more can be done, or in which immobilization alone is considered sufficient.

THORACOPLASTY

The last procedure for surgical collapse of the lung is paravertebral thoracoplasty performed according to the technique of Sauerbruch. It consists in resection of paravertebral segments of ribs, from the first to the eleventh inclusive. As Gravesen has shown, when segments of 10 to 15 cm. of each rib have been resected, the best results are obtained when this resection affects the paravertebral segments of the ribs and even includes the tips of the transverse processes. The degree of collapse depends upon the lengths of the resected segments. No sufficient collapse can be obtained unless the first rib is resected. In fact the ribs are suspended from the first through the intercostal muscles and the first from the cervical transverse apophyses through the scalene muscles. In well-performed thoracoplasties the three diameters of the chest are affected; the anteroposterior because of the decrease of the length of the costal arcs, the frontal and vertical because of the dropping of the ribs.

This operation can be performed in one stage. It is more conservative, however, to perform it in two or more stages. I think that in thoracoplasty, as in thyroidectomy for toxic goiter, the surgeon should adjust the operation to the resistance of the patient. When the pulse becomes rapid and the blood pressure falls the operation should be ended. It is better to do much less than a little more; and it certainly is better to cure the patient in several stages than to kill him in one. During the last decade, following these principles the mortality of this operation has dropped from 75 per cent as it was with the large operations performed by Friedrich to about 14 to 10 per cent and even less.

The general indications for thoracoplasty are fairly definite at present; unilateral caseous pneumonic cases, with positive sputum, afebrile, in relatively good condition, and in which a pneumothorax was impossible or ineffective; tendency to retraction of the diseased lung with displacement of the trachea, heart and diaphragm to the diseased side; weight stationary or increasing and rather moderate expectoration; these are the clinical symptoms which characterize good surgical risks. Patients running a slight fever, presenting moderate spreads in the collateral lung, and showing some degree of toxemia, constitute, according to the intensity of these signs, only fair or even bad surgical risks. In our services we consider fit for thoracoplasty all cases of unilateral caseous pneumonic tuberculosis after the caseous content of the lesions has sloughed out. Another factor of great importance which should be taken into consideration in the selection of cases is the resistance of the patient, which is not always in direct relation to its clinical appearance. Unfortunately we do not as yet possess a clinical method comparable to the "functional test of the kidney," for measuring the resistance of the patient. It seems *a priori* that the determination of oxygen utilization capacity of the patient at rest and exercise and the changes in the heart output could give us valuable information on the resistance of the patient and consequently of his ability to resist the operation. We are working on this question at the present time. In the meantime we consider that hemoglobin above 70 per cent, pulse below 100, little or only moderate increase in respiratory rate after a standard exercise, integrity of the kidneys and of the gastrointestinal tract are the requirements for the operability of the patient. On the other hand, tuberculous laryngitis, slight gastrointestinal tuberculosis, streaking and even hemoptyses do not constitute contraindications to operation.

The average results of thoracoplasty as compiled by the cases so far published in the literatures can be expressed as follows in round figures:

Without operation out of 100 patients with caseous pneumonic tuberculosis in which pneumothorax was not effective, 50 per cent died within a year, 30 per cent more within two years, 10 per cent more within three years and only 10 per cent survived over four years (Nissen). According to Barnes and Barnes, in 1465 cases with cavitations, the average span of life was 15.8 months, and 80 per cent died within the first year. Out of 100 similar patients submitted to thoracoplasty (Nissen) 50 were completely cured; that is, within one to two years after operation they were able to perform a full day's work without fever, cough or expectoration and were permanantly bacilli free. Twenty were alive ten years after operation, greatly improved, the most of the time bacilli free, without fever and able to perform some work. Fifteen to 20 died from the operation and in 10 the course of the disease had not been influenced by the operation. In other words of 100 patients doomed without thoracoplasty to certain death, obliged in the meantime to spend their life in sanatoria, and constituting a real danger for the community, 60 to 70 were able to return to active life and become an asset instead of a liability to society.

In this postoperative mortality are included all deaths occurring within three months after the operation when related to it. Ten to 20 per cent mortality may appear considerable. But if we consider that 100 cases represent 200 or 300 operations, upon patients the condition of whom is rather precarious, these figures become more acceptable. Furthermore I think that the surgeon must not try to correct his statistics by decreasing the extent of the operation. Surgeons who resect 1–3 cm. of ribs have no mortality, but they have no cures either. When we decide to operate upon a tuberculous patient we must perform a real thoracoplasty, that is the resected segments of the eleven ribs must have a total length of 160 to 180 cm. with at least 7 cm. of the first rib. Otherwise the operation is useless. Incomplete thoracoplasties discredit the operation more than 10 to 20 per cent mortality. That is probably why some authors, as Fishberg in his book, have stated that they never saw a tuberculous patient cured by a thoracoplasty. It is in tuberculosis as in cancer. Radical operation presents a higher mortality but also can show definite cures.

In my statistics of 72 cases I had a 16 per cent mortality. But on the other hand, 76 per cent of my patients became and remained bacilli free.

The principal causes of postoperative mortality are: the so-called aspiration pneumonias and bronchopneumonias, heart failures, mediastinal flutter, postoperative shock and infection of the wound. Wound infection can be avoided to a great extent with well-regulated team work and faultlessly functioning operating rooms. Occurrence of serious shock can be diminished by good selection of patients, adequate preoperative care, well-regulated operative technique, and skillfully conducted cardiotonic treatment, hydration of the patient and transfusions. Here again the close cooperation of the surgeon with the phthisiologist are of the greatest importance. Postoperative pulmonary complications and heart failure constitute the two most important causes of postoperative morbidity and mortality. I place them together because I believe that they have the same cause, which is acute or subacute but prolonged anoxemia. In fact according to the ideas I have expressed elsewhere with Dr. Birnbaum, and in favor of which we presented clinical and experimental evidence, I firmly believe that these postoperative complications are due to bronchial obstructions to the impairment of bronchial drainage, the production of atelectasis and the resulting marked impairment of the respiratory and circulatory functions. As a matter of fact we operate upon patients lying on the healthy side so that during

operation purulent exudate coming from the diseased lung necessarily drains into the bronchi of the other lobes and is gradually carried down into the smaller bronchi. The amount of sputum thus drained during the operation varies according to the amount of the expectoration of the patient and to the degree of surgical collapse produced in the diseased lung during the operation. If the patient is not well under anesthesia so that during operation he becomes restless and makes crying gestures he may be induced to take deep respirations and the infected fluid is carried farther down into the smaller bronchi. Thus, bronchial obstructions and atelectasis, massive or lobular, can start on the operating table or shortly after as in the cases reported by Berry. This is not all. During anesthesia the patient aspirates a considerable amount of mucus and saliva, coming from the mouth and rhinopharynx, which always contains aerobic, and anaerobic microorganisms and more particularly pneumococci group iv. Lemon and Higgins have shown that by simply dripping into the mouth of an anesthestized dog 20 per cent India ink solution, this is found in the small bronchi within forty minutes. Myerson has shown by bronchoscopic examination of patients undergoing tonsillectomy that blood is present in the bronchi in 90 per cent. Thus the three factors necessary for production of pulmonary complications, namely, bronchial stasis, impairment of bronchial drainage and increased viscosity of bronchial exudate, due to the pneumococcus, are present. Many cases of patchy or massive pneumococcic atelectasis rapidly change to bronchopneumonia or lobar pneumonia. In all our cases which presented such complications the sputum, carefully checked, always contained pneumococcus group iv. These complications lead to anoxemia, to shallow rapid breathing and to "acapnia" (washing out of CO_2). Thus a vicious circle is created. Acapnia will further increase anoxemia, because of the decrease of the physiological stimulant of the respiratory center (CO_2) and because of the Bohr effect, that is the higher saturation of hemoglobin with oxygen in the presence of lower tension of CO_2 and the formation of oxyhemoglobin in which oxygen holds fast to the hemoglobin and is not liberated in the tissues. If this condition is not rapidly relieved disorganization of the respiratory center will occur. The best way of relieving this condition is persistent inhalation of O_2-CO_2 7 to 10 per cent. This mixture is administered by intranasal catheter or better by placing the patient in an oxygen tent for twenty-four to forty-eight hours whenever symptoms of anoxemia-acapnia develop. In cases of atelectasis immediate bronchoscopic liberation of the bronchi is advised, if the obstruction is not rapidly relieved by moving the patient from side to side. Transfusion is of a great help because it increases the amount of circulating oxyhemoglobin and thus immediately corrects anoxemia.

The heart and the vasomotor centers, on the other hand, are no less sensitive to anoxemia than the respiratory center, so that fall of the blood pressure and dilation of the heart rapidly occur. I believe that the so-called heart failures are nothing else but acute dilatations brought about by anoxemic crisis. The practical conclusion to draw from the foregoing considerations is that it would be desirable to elaborate an operative method by which the following requirements could be fulfilled: (1) keep the patient during operation under quiet anesthesia by maintaining a constant pulmonary ventilation; (2) cut off as much as possible any communication between the upper and lower respiratory tract; (3) which is of the greatest importance, remove from the bronchi, before, during and at the end of the operation the exudate accummulated in them or drained into them from the diseased lung.

For these reasons, I actually use[1] general anesthesia with gas-oxygen and a small amount of ether, administered through a specially devised "intratracheal" catheter. This intratracheal catheter is introduced

1 *Anesth. & Anal.*, 11: 138, (May-June) 1932.

into the trachea under local anesthesia and the pharynx tightly packed with gauze. This procedure, when skillfully done, can be accomplished without the slightest inconvenience for the patient. The results so far obtained in 150 thoracoplastic operations performed upon 72 patients, have allowed us to eliminate pulmonary complications, and gave a surprising decrease in postoperative morbidity and in spread of the disease to the healthy lung.

In closing this study of surgical treatment of pulmonary tuberculosis, I shall mention two procedures, namely, pneumonostomy and pneumonectomy.

Pneumonostomy is the opening and draining of tuberculous cavities through the chest wall as in putrid abcesses of the lung. Tuberculous cavities have been accidentally or deliberately opened and drained. Dr. Lilienthal reported 3 similar cases, and in a few cases good results have been obtained. In our service however we were not successful with this procedure.

Pneumonectomy is the resection of the tuberculous lung, as in bronchiectasis. MacEwen had a striking result in one case, in which he performed, in several stages, an almost complete resection of one lung. This procedure is indicated in cases of tuberculosis in which bronchiectasis has secondarily developed, and more particularly when this occurs after otherwise successful thoracoplastic collapse.

PLEURAL COMPLICATIONS

When serous fluid is present or purulent exudate containing only tubercle bacilli, unless it is threatening the life of the patient because of its amount, the best method of treatment is to avoid any repeated tappings which often are followed by external fistulae and secondary infection of the exudate by pyogenic germs. The treatment of choice of tuberculous empyema is thoracoplastic collapse of the pleural cavity.

The same is not the case when, following spontaneous pneumothorax or fistula of the thoracic wall, mixed infection results. According to the degree of infection, oleothorax with gomenol oil or injection of dyes may be sufficient for checking the infection. If these do not prove to be successful then the cavity should be washed and drained, preferably by the closed method. As soon as the acute infection is over, if the lung does nor or should not be allowed to expand, a thoracoplasty should be performed in as many stages as the condition of the patient indicates. In cases of putrid empyema immediate drainage must be performed. The prognosis in septic cases is very serious and in putrid empyema almost always fatal.

Early diagnosis of spontaneous pneumothorax is therefore important. Generally the clinical symptoms are sufficient for the establishment of the diagnosis, and injection of gentian violet in the pleural cavity allows the disclosure of the bronchial fistula. But it is not always so. Sometimes it is difficult to arrive at a definite diagnosis of the presence of a pleuropulmonary communication. It might be of interest, therefore, to mention a method developed in our services for the diagnosis of these cases.[1] It consists in the analysis of the gaseous contents of the pleural cavity. Whenever the oxygen is less than 2 per cent and CO_2 more than 7 per cent spontaneous pneumothorax with persistent bronchial fistula can be excluded. The same method allows the measurement of air contained in the chest, and by successive readings during refillings, the determination of the absorbing capacity of the pleural serosa for O_2 and CO_2.

CONCLUSIONS

1. By surgical treatment of tuberculosis we must understand a number of operative procedures which are able to immobilize, collapse and compress the lung, in order to collapse the cavities and help cure the patient.

2. The adoption of a standard classification of the disease is necessary, so that indications, results and statistics may be comparable and of real scientific value.

[1] *Am. Rev. Tuber.*, 26, (Aug.) 1932.

3. Collapse therapy is indicated only in the caseous form of tuberculosis, and only after the period of consolidation is over when the necrotic material has been sloughed out.

4. The widespread idea, expressed by Dumarest under the epigramatic sentence "pneumothorax is the treatment of the acute period of tuberculosis and thoracoplasty of its chronic phase" is not completely correct in the first part. In the very acute period of tuberculosis any collapse therapy is useless, because of the impossibility of collapsing a consolidated lung and because exudative tuberculosis generally heals by itself; it is dangerous because of the frequency of the occurrence of spontaneous pneumothorax.

5. Pneumothorax if it does not produce selective collapse is useless and harmful and should be replaced by more efficient collapse therapy.

6. Phrenicectomy is unable by itself to produce a definite cure of tuberculous cavities. It is useless in the other forms of tuberculosis. Associated with other collapse methods it may be of real help.

7. Intrapleural pneumolysis by the method of Jacobaeus, in well-selected cases and skillfully performed, can give good results.

8. Extrapleural apicolysis with or without compression of the apex by foreign bodies as packing or "plumbe" is a method which is especially indicated in bilateral cases.

9. Thoracoplasty is the most efficient method for permanent and definite collapse of the lung. It is, however, a major operation, requiring a faultless technique, a careful selection of cases, adequate preoperative preparation and painstaking previous estimation of the resistance of the patient; ether anesthesia is harmless, but should be administered by such methods as to allow a complete separation of upper and lower respiratory tract and permit the suction of the bronchial exudate. Intratracheal anesthesia fulfills these requirements. The most important postoperative complications are cardiopulmonary complications generally following bronchial or bronchiolar obstruction and the resulting atelectasis and anoxemia.

10. The treatment of pleural complications varies according to the nature of the exudate; it should be conservative, in aseptic tuberculous exudates. Thoracoplasty is the treatment of choice. Injection of gomenol oil or dyes is to be tried in moderately infected empyemas; drainage should be installed in mixed infections and more especially in anaerobic empyemas later followed by thoracoplasty.

11. Thoracic surgery in tuberculosis requires the close cooperation of the thoracic surgeon with the phthsiologist, not only before but also after operations; tuberculosis patients should no more be considered as medical or as surgical patients, but as medicosurgical cases requiring continuous medicosurgical supervision, study and treatment.

Pulmonary tuberculosis can be successfully treated "by mechanical means" as Carson prophesied. This part of the problem is solved. What remains to be done is to perfect our technique and to devise some means by which mortality will be decreased and operative indications enlarged.

In closing I wish to quote the wise words of Fichel. We should not forget, says this author, in studying the treatment of cavities, that:

> Time is an important factor in the treatment of pulmonary tuberculosis and that all indications are strictly limited by time and hold good only for the short duration of the respective stage of the disease. After all, treatment of tuberculosis is the treatment of tuberculous cavities. Opportunities are quickly missed and chances for more conservative measures easily overlooked until finally the most radical procedures have to be resorted to. In going over the histories and films of chronic patients one can read many tales of "what might have been" and cannot help thinking that there is perhaps a period, however short, in the life of every tuberculous patient when the closure of his cavities by the appropriate method may turn the scales in his favor.

FRACTURE OF SESAMOID BONES*

A REPORT OF TWO CASES

JACOB SAGEL, M.D.

MINNEAPOLIS, MINN.

FRACTURE of the sesamoid bones is a relatively rare condition and very little on this subject is reported in the literature. The cases reported are usually fractures of the sesamoid bones about the great toe, but these rarely occur without fracture of other bones of the foot. I have seen no case reports of fracture of the fabella, the sesamoid bone located in the head of the lateral tendon of the gastrocnemius muscle. This report consists of 2 cases; one a fracture of the fabella and the other a fracture of the external sesamoid of the great toe unassociated with fracture of any other bones of the foot.



FIG. 1. Intercondylar fracture of left femur. Note fracture of fabella (arrow) with separation into two large fragments and several smaller ones. Deformity of head of tibia and fibula is due to previous fracture.

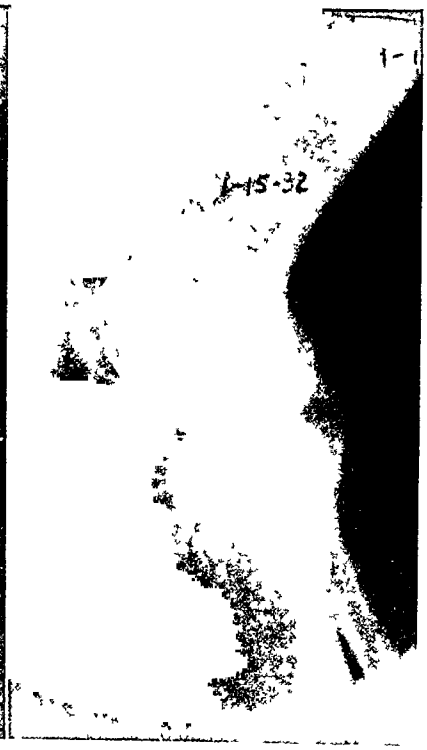


FIG. 2. Same case, twenty-seven months later. Union of two large fragments of fabella has occurred.

CASE 1. A carpenter, sixty-seven years old, injured his left knee October 2, 1929, sustaining an intercondylar fracture of the left femur and a stellate fracture of the fabella, an inconstant sesamoid bone in the external tendon of the gastrocnemius muscle at the level of the popliteal space. To ascertain the exact mode of occurrence of the fabellar fracture, I questioned the patient as to the details of the injury. He was reaching for a small board which lay behind a pile of plaster boards, each 4 by 12 feet, when the pile fell

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Presented before the Minnesota Radiological Society, Feb. 13, 1932.

against him. He was thrown upon a beam which was lying across a roll of insulating felt so that his left knee was caught between them in a partly flexed position. Following the injury he remained in the hospital for three weeks. At present he has almost no trouble in walking.

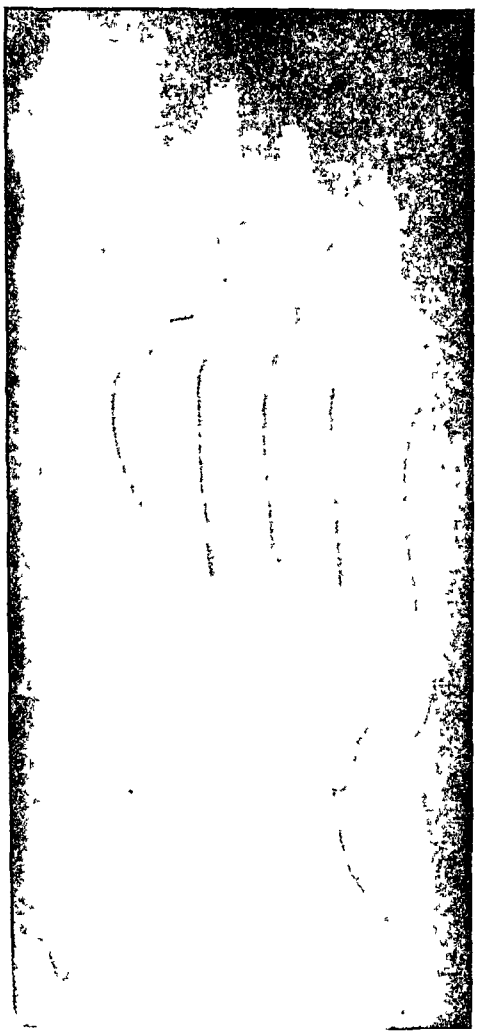


Fig. 3. Congenital bifid division of fibular sesamoid of great toe. Note smooth, rounded contours of two parts, which are in close apposition, and transverse line of division, with no evidence of union. Compare with Figure 4.

The first examination (Fig. 1) shows the intercondylar fracture of the left femur and the stellate fracture of the fabella. Multiple irregular fragments of the fabella, which are somewhat separated, are shown. The appearance is characteristic of a fracture of a sesamoid bone and does not at all resemble the appearance of a bifid sesamoid which has smooth rounded edges with the two halves in close apposition (see Fig. 3). Figure 2 shows the appearance on January 15, 1932, at which time union of the larger fragments had occurred.

Case II. A man, aged forty, was injured in an auto accident, sustaining a crushing fracture of the external sesamoid of the left great toe (see Fig. 4). One large fragment and several smaller ones are shown. It is of interest that none of the other bones of the foot was fractured. The foot was caught between the floor of the car and the door thus crushed the external sesamoid of the great toe by direct trauma. Follow-up examination on this patient could not be obtained.

The chief diagnostic importance of this fracture is to differentiate it from a congenital division of the sesamoid bones, which is not uncommon (Fig. 3). Division of the tibial sesamoid of the great toe is more common than that of the fibular sesamoid. Burman and Lapidus[1] report 6 cases of division of the fibular sesamoid and 72 cases of division of the tibial sesamoid of the great toe in a series of 1000 cases. A. H. Bizarro,[2] in his review of the literature, noted only 6 cases of fracture of the fibular sesamoid of the great toe. The case reported here, a fracture of the fibular sesamoid of the great toe, without fracture of any other bones of the foot, is very rare. In congenital division of the sesamoid bones the two parts have smooth contours, generally show a transverse line of division, and are usually in close apposition, whereas the fragments of a fractured sesamoid usually have irregular contours and may be widely separated. Burman and Lapidus state that callus unites the ends of a fractured sesamoid bone which then definitely distinguishes it from a congenitally divided one. Union occurred in our case of fractured fabella (see Fig. 2). A comparison of all of the plates shown demonstrates well the difference between these two conditions

[1] *Arch. Surg.*, 22: 936, 1931.
[2] *Ann. Surg.*, 74: 783, 1921.

so that no difficulty should be encountered in making the diagnosis.

The rarity of fractured sesamoid bones should make one hesitate somewhat in diagnosing this condition unless the definite findings shown here are present.

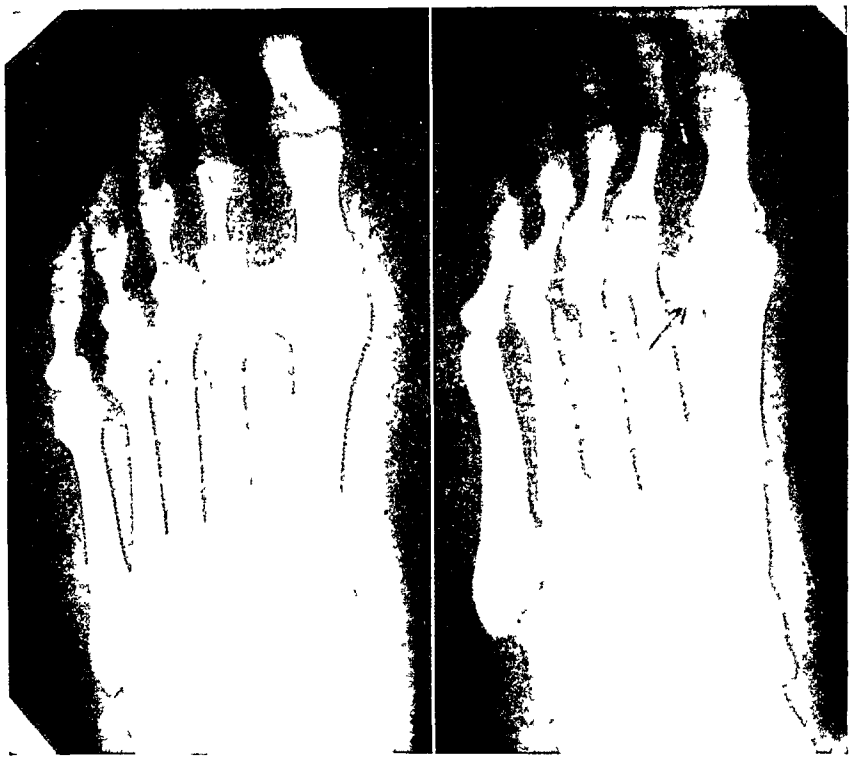


Fig. 4. Fracture of fibular sesamoid of left great toe. One large fragment and several smaller ones, all with irregular edges are shown. Note absence of fracture in other bones of foot. Compare with Figure 3.

THE WRIGHT METHOD OF TREATING LEG ULCERS*

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DURING my internship in the Charity Hospital in New Orleans, twenty-eight years ago, a valuable lesson was learned concerning the healing of chronic leg ulcers. An old street sweeper had been coming to the out patient clinic two or three times a week for twenty years to have a varicose leg ulcer treated and dressed. The custom then was to change the attending resident every six months, so during this time he had been treated by at least forty doctors and no doubt had almost as many different kinds of treatment applied. The ulcer remained unhealed. At this time he was knocked down by a runaway horse and sustained a fracture of the thigh. It was necessary to put him to bed for ten or twelve weeks to treat the fracture; the ulcer healed while he was in bed with no special medication.

The common treatment for this condition thirty years ago was to apply various kinds of salves and medicines, dress the leg every few days and give mixed treatment. In the mind of the attending surgeon the patient was regarded as a syphilitic until proven innocent. The presence or absence of syphilis has little effect on the healing of the ordinary leg ulcer. Syphilis as an etiological factor enters into the causation of chronic leg ulcers in a remarkably small percentage of cases.

The use of various antiseptics has had little effect on chronic leg ulcers. The failure to heal is due to venous stasis and poor return circulation. This fact is generally conceded now.

Leg ulcers are most certainly not as commonly encountered now as they were in the first decade of this century. There has been in the past twenty years considerable advance in the treatment. Beck's adhesive plaster strapping of the ulcer was a valuable contribution. The practice of surgical removal of the varicose veins, and more recently of injecting irritating solutions to block the veins, has caused many chronic cases to heal. The use of the supporting boot of Unna's paste is a very useful method of treatment.

Notwithstanding these advances a goodly number of cases of chronic leg ulcer are still to be found in the out patient departments of our large hospitals. This is a condition that occurs much more commonly among the poor. In the rural districts more cases are present than the average physician would think. These people, on account of poverty and discouragement from past futile treatment, do not come to the doctor. The sum total of their suffering and disability is still great.

On several occasions during the past few years Mr. Dickson Wright of St. Mary's Hospital, London, has described a method of treatment which is most efficacious. He read a paper on this subject before the British Medical Association in Winnipeg, in August 1930, and I had an opportunity to see him demonstrate his method during the following month. Since then I have had occasion to treat 23 cases of leg ulcer by his method. The principle underlying Mr. Wright's method of treatment is that of compressing the leg with elastic adhesive plaster, thereby overcoming the passive congestion due to the diseased and dilated veins.

Over one hundred years ago Thomas Baynton, a surgeon of Bristol, made the following observation: "This disease is due to the unfavorable situation of the parts which are placed at a remote distance from the fountain of life and heat, and are obliged to return the venous

* Read at the meeting of The Southern Surgical Association, White Sulphur Springs, W. Va., Dec. 10, 1931.

blood and lymph to the heart under peculiarly unfavorable and disadvantageous circumstances." Mr. Wright states:

A simple experiment proves his contention: A needle connected to a glass tube 8 feet long is inserted into any varicose vein, the blood rises instantly to the level of the ears; if he now lifts a heavy weight in both hands the upper level is now far above his head and the venous pressure even greater than the arterial. Put the hydraulics right and the ulcer will take care of itself is the gist of the method. The edema is harmful and the varicose circulation useless, so they should be squeezed out of existence by an adequate and uniform compression.

The technique of Mr. Wright's method of treatment is as follows: When the patient first presents himself with a chronic leg ulcer, if there are visible varicose veins, one of these rather high up in the leg is injected with 5 per cent sodium morrhuate solution. I have used this solution and quinine and urethane and find the sodium morrhuate is more effective. Immediately after the injection of a varicose vein an elastic adhesive plaster bandage is wound snugly around the leg from the foot or ankle to just below the knee.

In the first week or so of the treatment if the ulcer is foul with a very free discharge, it will probably be wise to change this dressing twice a week. Later the plaster is usually left on for a week and in the late stage of the treatment, often longer. If the ulcer is painful, there may be a little more pain for the first few hours after the application of the plaster, however, by next morning the pain is all relieved and does not return during the course of the treatment. Within ten days or two weeks a large ulcer, which has resisted previous treatment for years, shows great evidence of healing and the granulations, which have been indolent and unhealthy, become pink and firm and show every evidence of a healthy surface with rapid epithelization at the edges. One vein is injected at each subsequent application of the plaster. The injection of the veins is not at all necessary to cause the healing of the ulcer under the plaster, but this is a convenient time to inject the veins to prevent a recurrence of the ulcer. This method is simplicity itself and can be applied by any physician.

In his paper before the British Medical Association Mr. Wright reported 324 cases treated with 321 healed. Two discontinued treatment for some cause and one patient died of abdominal cancer before finishing treatment. Mr. Wright further states:

The advantages of the compression are as follows: (1) It abolishes the varicose circulation. (2) It diminishes the oedema, in some cases reducing the volume of the leg by 4 or 5 pints. (3) It thus reduces the girth of the leg in oedematous cases, and thereby reduces the width of the ulcer by the same amount before any healing takes place at all. (4) It approximates the edges of the ulcer. (5) It protects new epithelium and delicate granulations from dressing trauma. The discharge lifts the sticking plaster away from the ulcer, and renders removal painless and harmless to the epithelium and granulations. (6) It presses down and softens the raised margins of an indurated ulcer, so rendering it flat instead of excavated. (7) It provides a "pansement specifique." (8) It abolishes pain in the majority of cases. (9) It permits full functional activity, and patients who work and take exercise are cured more quickly than those in bed. (10) It saves the expense of dressings and lotions. (11) It requires no particular skill. (12) In early ulcers (up to six months' duration) there are numerous invisible islets of epithelium buried in the granulations; pressure brings these to the surface and they quickly cover the ulcer. Frequently an ulcer of the size of the palm of the hand, if of short duration, will heal within seven days. (13) It cleans the ulcer more quickly than any antiseptic method, and the foetor rapidly disappears. (14) It brings to the surface varicose veins which were deeply buried in oedema, thus rendering injections possible which otherwise could not be attempted. (15) It gives a supple scar, which loses its adherence to the underlying bones.

He further states:

Six years' experience with this treatment enables me to promise the patient: (1) To

cure any ulcer, no matter of how long standing, or to what extent adherent to bone, at the rate of one square inch per week. (2) To relieve all pain. (3) To allow full work and exercise. (4) To eliminate the possibility of recurrence in nearly every case.

To the uninitiated in this method of treatment these claims must sound most extravagant. The experience which I have had with 23 cases leads me to accept these claims with practically no mental reservations. In addition to being a specific treatment for varicose leg ulcers I have found the treatment very efficacious in other leg ulcers. During the past winter a colored girl, aged nineteen, was referred to me with a leg ulcer 2 inches by 3 inches in diameter, on the left leg. This ulcer had been present for nine years. On examination it was immediately discovered that she was somewhat anemic and jaundiced and in reply to questions she gave a history of at times having cramps in her abdomen and some joint pains. This, of course, suggested sickle-cell anemia as the cause of the ulcer. Blood examination confirmed this suspicion. It was explained to her that certain cases of sickle-cell anemia could be cured by splenectomy. In her case an incision was made for splenectomy. The spleen was, however, found atrophied so that it was no larger than two joints of the thumb. These cases with extreme atrophy of the spleen apparently are not benefited by splenectomy. The surgeon being aware of this fact and also due to the fact that she was extremely tall, 6 feet, with a very long chest, so that the spleen was placed very high above the edge of the ribs, it was judged there would be some slight danger in removing this spleen with the probability of no benefit, so the abdomen was closed without splenectomy. She was kept in bed for two or three weeks for the wound to heal. During this time there was only slight evidence of healing of the leg ulcer although she was quiet in bed. After she was up and about, the plaster was applied in the usual manner and the ulcer healed rapidly and firmly in five weeks. In this case there was no evidence at all of venous stasis, in the ordinary acceptance of the term. This girl, although she was 6 feet tall, weighed only 107 pounds and the circumference of the calf of her leg was only 9½ inches.

Mr. Wright has found that this method of treatment does well with almost every kind of ulcer, such as bed sores, perforative ulcers or denuded areas after amputation of the breast.

Sir Almoth Wright has made certain experiments studying the reasons for the benefit in ulcers not due to varicose veins. In an untreated ulcer a cover slide is laid on the ulcer and becomes coated with discharge and then is laid wet side down on a petri dish and cultured. The result shows colonies everywhere: B. coli, staphylococcus, streptococcus, B. pyocyaneus, etc. In a treated ulcer the same is done. Colonies now appear not under the cover glass at all but only where the fluid has dried beyond the edges of the cover glass. The inspissation kills the leucocytes or bacteriophage or whatever it is and the organisms live.

This elastic bandage is really practically an ACE bandage with zinc oxide adhesive applied to one side of it. After the plaster is applied there is a free discharge of serum through the mesh of the plaster. Certainly some antiseptic or chemical action takes place on the bacteria on the surface of the ulcer which has a most favorable action on the healing process. The wound is dressed with its own discharge, the "pansement specifique" of Besredka.

In the cases of large ulcers the time of healing can be considerably reduced by skin grafts. After the plaster has been applied for two or three weeks the granulating surface becomes pink and healthy with the skin edge on the lever with the granulation. Almost any kind of properly applied skin graft will take on this surface, with the patient up and about and the plaster placed over the ulcer and the skin graft, in the usual manner. We have made this a simple office procedure, removing under local anesthesia pieces of whole thickness skin ½ inch in diameter, from the patient's thigh and placing them around

over the ulcer. Within ten days they have firmly taken and new areas of granulation develop from them. Mr. Wright commonly uses a method of darning strips of skin under the granulation surface with a needle. This method of grafting is very useful and was described by Dr. William L. Keller of Washington.[1] Mr. Wright has recently fired pieces of skin into the ulcer with an air pistol and had them take.

The greatest advantage of this method of treatment is that it is just as efficacious in the leg ulcer following a postoperative or postinfection phlebitis as it is in the ordinary varicose ulcer. The method of application is exactly the same. It is surprising how rapidly these ulcers will heal under the elastic adhesive plaster.

J. B., colored male, aged sixty, first seen November 4, 1930.

Twenty years ago he had typhoid fever followed by a phlebitis, the leg swelling and giving him a great deal of trouble since then. Thirty-three years ago he had syphilis. Nine years ago he developed an ulcer on the left leg.

When first examined the calf of the right leg measured 16 inches and that of the left leg 19 inches. The left leg was greatly swollen and there was a large ulcerated area on the lower external surface of this leg and a smaller one on the inner surface. The large ulcer was 3 inches by 2 inches.

The plaster was applied in the usual manner. After the edema was squeezed out by the plaster a few varicose veins were discovered and injected at one week intervals. This ulcer was completely healed by the middle of January, 1931. No specific treatment was given as his Wassermann reaction was negative. On discharge the circumference of the left leg was 15 inches where it had been 19 inches at the beginning of treatment. This ulcer had resisted other forms of treatment for nine years and was completely healed under this treatment in nine weeks.

Mr. Wright has reached the conclusions that the average rate of healing of the ulcer under the elastic bandage is one square inch per week. Some of the recent ulcers heal much more rapidly.

We had one case in the person of a colored cook whose ulcer had been present only eighteen months. His ulcer was so painful that he frequently had to call a doctor at night to give him morphine, and often he had to use two crutches in walking. He came to our office with one crutch and a walking stick to assist him in walking and he was suffering greatly from the leg. He had suffered with phlebitis two years before and the ulcer had been present for eighteen months. Soon after the development of the ulcer some physician gave him *x*-ray treatment. The pain and discomfort from the ulcer were so great that we feared he might have an *x*-ray burn. After applying the plaster the pain was relieved in sixteen hours, and the ulcer entirely healed in two weeks. In this case there were areas of skin at various places over the ulcer which could not be seen until after the plaster was applied and removed at the end of a week.

Mr. Wright reports one remarkable case:

James W. age fifty-two, thirty years ago in the Boer war developed typhoid fever complicated by femoral thrombosis and an ulcer developed immediately after he got about; since then this has never healed but has slowly grown to encircle the leg and measures 31 square inches in area although his total hospitalisation over the thirty years amounted to 7½ years. Every treatment including skin grafting has been carried out, amputation had been repeatedly refused and the Pension Committee had awarded him a lifelong pension of two pounds a week. A most conservative estimate of the cost of this ulcer to the patient, and state gave a figure of over five thousand pounds! With 21 weeks of the treatment outlined above he was completely cured and during the whole period of treatment followed the most unfavorable of occupations, viz. that of a house painter, earning four to five pounds per week. He stated that during the treatment he had less pain than at any time in the course of the disease. There were no varicose veins at all in this case so the patient is now wearing an elastic support to prevent recurrence.

He states: "This treatment is just as diagnostic as 606 in syphilis, if an ulcer does not heal it is either cancer, syphilis or an *x*-ray burn, if a treatment is diagnostic, it is specific, effective."

After fifteen months' experience with this method of treating chronic leg ulcers I am of the firm opinion that Mr. Wright's method of treatment is a very valuable contribution, and his claims not overstated.

[1] *Ann. Surg.*, June 1930.

DIFFICULT HERNIAS

SURGICAL TREATMENT WITH SPECIAL REFERENCE TO A MODIFIED METHOD OF OBTAINING LIVING FASCIAL SUTURES*

JOSEPH E. FULD, M.D.

NEW YORK CITY

THE advent of living fascial sutures is a distinct advance in the operative treatment of certain forms of hernias.

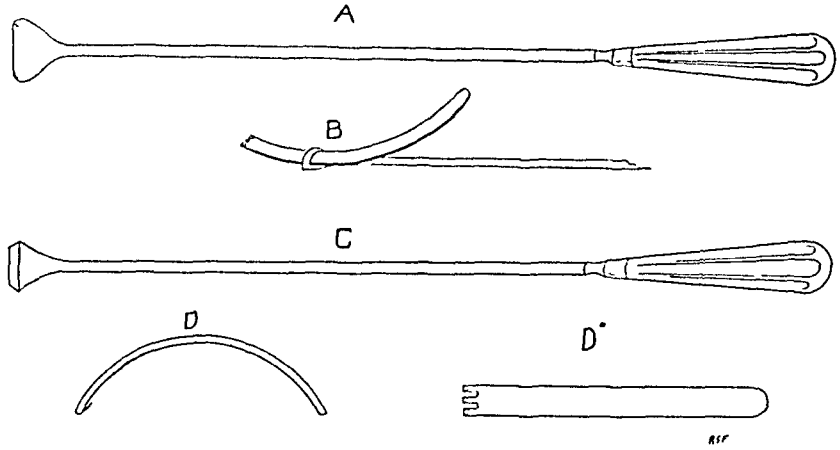


FIG. 1. A. Fuld's fascia separator. B. Fuld's fascia threader (in position). C. Grace's fascia stripper. D. 1. Fuld's fascia threader (side view). 2. Fuld's fascia threader (top view).

Before the introduction of living fascial sutures by Gallie and Le Mesurier in 1923, there was considerable confusion as to what was the best operation for large ventral, recurrent, direct and inguinal hernias.

When other than living suture material is employed in hernia repair, the postoperative success is dependent upon the process of healing by delicate scar tissue. The scar is not strong and slowly stretches when subjected to strain. The success of the living fascial suture method is based on the fact that the structures of real strength, the musculo-aponeurotic borders of the ring are left strong enough to withstand considerable strain. Further factors in the success of fascial suturing are strength and permanence of the sutures used. These sutures neither stretch nor contract. Due to the well-known fact that fascia lata transplanted continues to live practically unchanged after transplantation, the hernial defect becomes permanently closed and the hernia remains cured.

The principle of using the living fascial sutures differs from the use of other sutures in that other sutures draw the conjoined tendon and Poupart's ligament together, or draw together the margin of the ring in a ventral hernia with the hope of securing edge-to-edge apposition and union, while the living fascial suture is woven or laced into the space between the conjoined tendon and Poupart's ligament, thereby closing the deficiency of Hesselbach's triangle in a direct hernia, or by the process of basket-like weaving similar to the manner in which one would close the hole in darning a sock. The closure does not depend upon drawing together the weakened and atrophied edges under tension, but by filling in the intervening space with a filigree formed of the fascial inlay the ventral hernia may be closed.

*Read before the Clinical Society of the Correction Hospital, New York City, Oct. 27, 1932.

When a defect is present and support is required, as is usually the case in difficult hernias, no other material can be used with such satisfactory results. The Gallie operation, which is admittedly superior to older operative methods for the cure of hernia, is, however, frequently refused by the patients because of the long scar which results when fascia is taken from the thigh. The Gallie operation is also accompanied by an increased period of disability, due to operation on the thigh or by infection of the thigh wound in some cases.

These objections have been largely overcome through the use of the fascial stripper devised by R. V. Grace. Because of the density of the connective tissue, considerable technical difficulty is frequently encountered in removing the fascial tissue with this stripper.

My modification of the technique suggested by Grace consists of using two additional instruments, which I have devised. (Fig. 1.) I am now able to remove the fascia more easily and more quickly with the Grace stripper. In a series of 35 cases in the past 6 months, I have been much impressed with the ease of technique when using these two additional instruments.

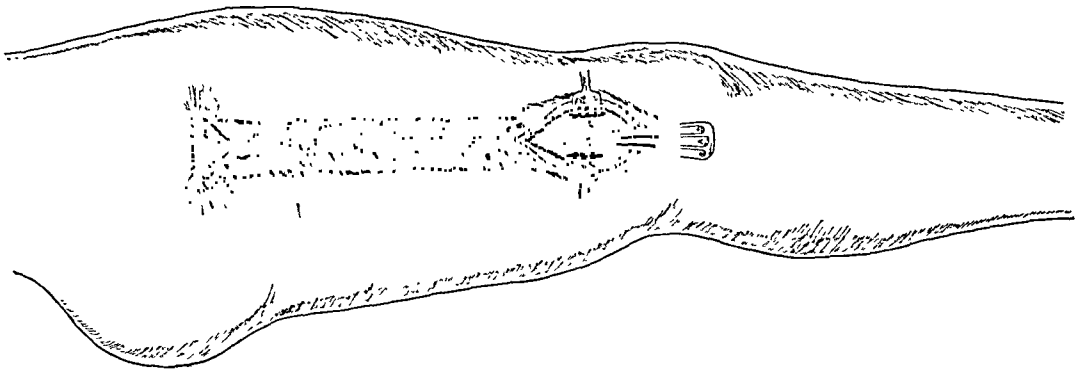


Fig. 2. First step. Separation of skin and connective tissue from fascia (instrument in situ).

TECHNIQUE

1. Make a 2 inch longitudinal incision on the outer aspect of the thigh beginning at the level of the upper border of the patella. (I prefer a longitudinal incision because the direction of the fibers of the fascia is more easily seen.)

2. Carry the incision to the fascia and dissect the skin and subcutaneous tissue laterally about an inch on each side of the incision, exposing clean fascia to view. Continue the separation at the upper angle of the wound for about 3 inches by blunt finger dissection.

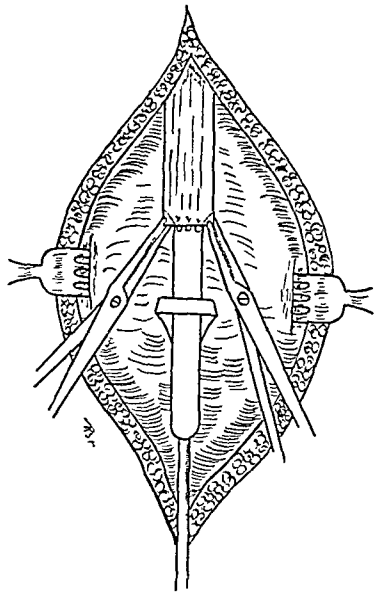


Fig. 3. Showing corners of fascia strip to be cut off to permit easy threading through stripper. It is very important to depress gripper end of fascial threader as it passes through stripper.

3. Separate the subcutaneous tissue from the strip of fascia which is to be removed. This is accomplished with my "fascial separator" which is constructed as a large size fascia stripper but differs from it by having the cutting end replaced by a

solid piece, with a blunt non-cutting end. This instrument is held at an angle of 20 degrees with the thigh, and is pushed upward to the full length of the thigh along the direction of the fibers of the fascia in the plane of cleavage between the latter and the subcutaneous tissue. (Fig. 2.)

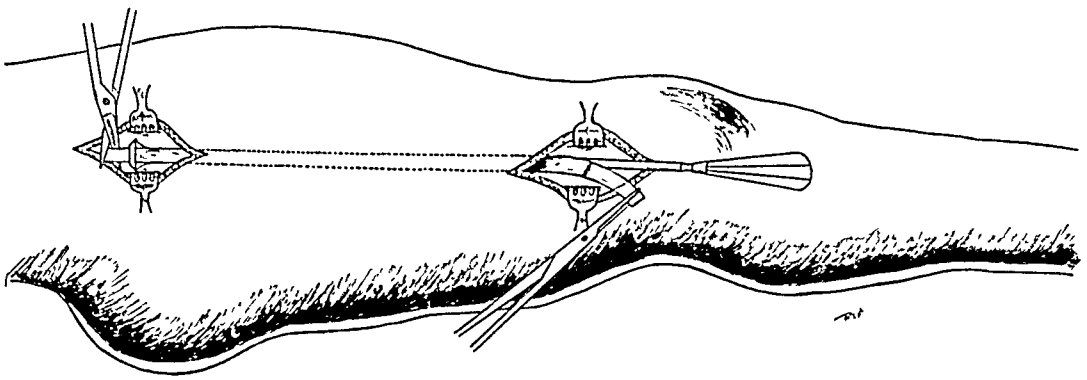


Fig. 4. Stripper method of obtaining fascia suture (final step).

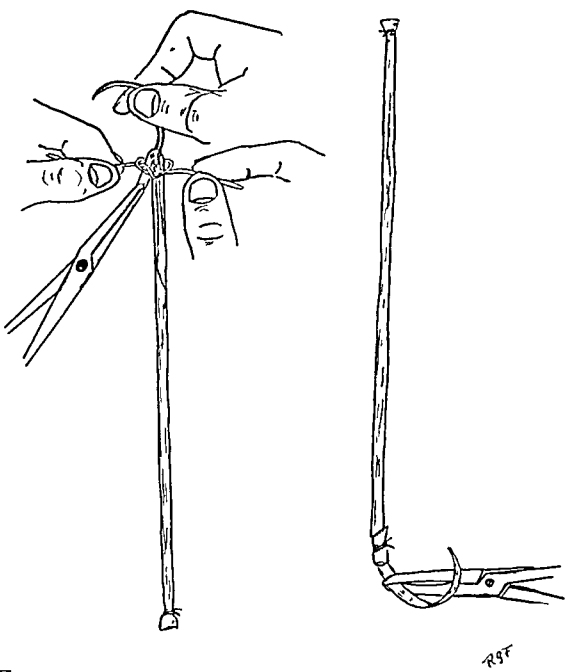


Fig. 5. A. Method of threading Gallie needle. A strip of fascia is tied securely into Gallie needle with silk, to prevent unthreading and a ligature of silk is tied around terminal end to prevent it from splitting. B. Fascial suture ready for use.

4. Now incise a strip of fascia in the line of fibers equal to the width of the stripper and cut it transversely at the lower end where the fascia is always thicker.

5. The strip of fascia is easily threaded in the stripper with the aid of my fascial threader. The threader is a thin, half-curved steel band, ½ inch wide and 4 inches long and has three small teeth ¼ inch from its end. The teeth are engaged in the substance of the cut fascia at the lower thickened end of the strip, which is now threaded into the lumen of the stripper. (Fig. 3.)

6. Remove the fascial threader and grasp the free end of the strip with a Kocher forceps. Push the stripper upward, holding the Kocher forceps taut. (Fig. 4.)

7. To cut the upper end of the fascial strip, incise the skin and subcutaneous tissue longitudinally in the upper part of the thigh where the stripper is visualized by a bulge. The stripper is then exposed and the attached end of the fascial strip is cut off. The two skin incisions are closed with interrupted silk sutures and a compression bandage applied.

8. After the fascia has been removed from the body it is cut (preferably with scissors) into strips ¼ inch wide. To save time in threading the needle it is advisable to use a Gallie needle which has a very large eye. The needle is threaded and the strip of fascia is tied securely into the needle with silk, to prevent unthreading. A ligature of silk is tied around the terminal end of the suture to prevent it from splitting when drawn through the tissues.

Gallie Operation: The general plan of the Gallie operation resembles that of the Bassini repair.

Technique of the Insertion of Living Sutures in Inguinal Hernia: The following is a brief description of the technique which

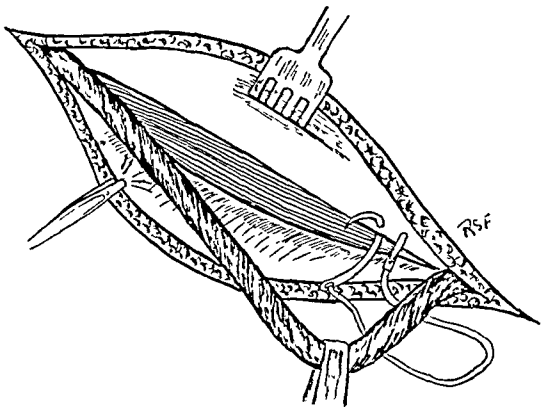


Fig. 6. Beginning of first step of basket weaving method (inguinal hernia). Needle and suture pass through Poupart's ligament from anchored end in sheath of rectus muscle and conjoined tendon.

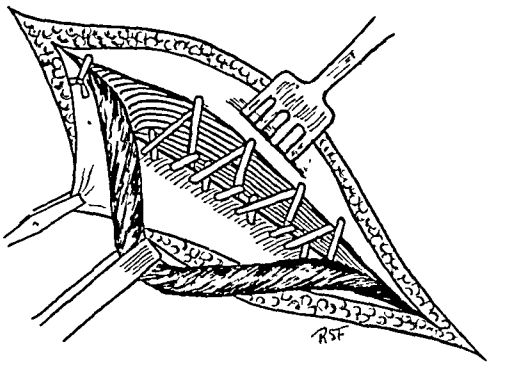


Fig. 7. Basket weave suture method (inguinal hernia). Insertion of second row of sutures. These are fixed above by a deep bite into line of junction of external oblique muscle and external oblique aponeurosis and placed close together.

I have adopted. It has given me excellent results.

1. The usual skin incision 4 inches long is made parallel to Poupart's ligament about ½-inch above. The spine of the pubis is exposed to the lower end of the incision.

2. The aponeurosis of the external oblique is slit up its full length in the direction of the fibres.

3. The aponeurosis is dissected well back on the inner side towards the rectus muscle, giving a full exposure of the internal oblique muscle.

On the outside the aponeurosis is dissected until Poupart's ligament is exposed to the pubic bone.

4. The sac and cremaster muscle are seized with a pair of thumb forceps and lifted up, and the muscular fibers are pushed aside by means of another pair of thumb forceps, thus exposing the sac plus the cord which are held together by the infundibuliform fascia.

5. The next step is to separate the infundibuliform fascia which surrounds the sac and cord in common, grasp the exposed sac with a clamp and separate the cord from the sac.

6. The sac is opened. The proximal end is dissected upward until it widens out in the general peritoneal cavity.

7. The sac is transfixed high up with a No. 1 plane catgut ligature and tied off, so that there is no longer any funicular process of peritoneum.

8. The cord is then held up by means of a piece of tape and artery forceps.

9. The fascial sutures are then transferred to the hernia wound and the suturing commenced. (Fig. 5.)

10. The anchoring stitch is passed through the anterior layer of the rectus sheath and rectus muscle close to their attachment at the pubic bone. From here it crosses the gap in the abdominal wall to the pubic spine, where it is securely fixed into the periosteum of the termination of Poupart's ligament. In this way a strong support is provided at the place where the hernia is most apt to occur. (Fig. 6.)

The suture is then continued in an outward direction to the reflected portion of Poupart's ligament until the internal ring is reached, thus closing the edge of the internal oblique muscle. Here the suture is knotted and then passed to the outer side of the cord, so that a new ring of fascia lata is formed. This stitch was first suggested by Coley.

By this means the weak spot produced by the passage of the cord through the abdominal wall is adequately supported.

In some cases a single line of sutures might be sufficient to establish a cure, but in the majority of patients, with a direct hernia the abdominal muscles are so weak that they cannot be depended upon to resist severe intra-abdominal pressure. (Fig. 7.)

To prevent the possible stretching of the internal oblique muscle, therefore, we insert a second line of sutures, superimposed upon the first, which passes from the strong abdominal aponeurosis at the junction of the external and internal oblique muscle and from the anterior sheath of the rectus muscle to Poupart's ligament. No attempt is made to draw these aponeurotic structures tightly together as the sutures are simply drawn sufficiently taut to make them lie flat. They are placed close together and made to pass under or through the first line of sutures, as in weaving a basket. In this way the weak spot in the abdominal wall is completely and permanently covered with muscle and strong fascia, and if care is taken in securing good anchorage for the sutures, there will appear no possibility of a hernia ever pushing its way through.

11. The cord is now dropped back into its new bed and the external oblique aponeurosis closed over it with a No. 1 chromic catgut suture and the skin wound is closed with interrupted silk sutures.

FEMORAL HERNIA

Recurrence is very common after operation for large femoral hernia, due mainly to ineffectual closure of the hernial opening. The usual operation consists of high ligation and excision of the sac, after which the recurved portion of Poupart's ligament is sutured to the pectineus muscle with kangaroo tendon or chromic catgut. Recurrence is due to two factors: first the constant pull exerted on the sutures by the muscle of the thigh and abdomen, and second the unsatisfactory union that is common when muscle is sutured to fascia.

If a securely anchored, properly placed living fascial suture is used the latter will remain in situ indefinitely and a permanent cure can be expected in every case.

Technique. Make an oblique incision 3 inches long parallel to and below Poupart's ligament directly over the hernial opening. Deepen the incision to the fascia lata of the thigh and expose Poupart's ligament, the falciform process of the fascia lata, the pectineus muscle and the neck of the sac.

A small firm pad of fat is always pushed through the femoral canal by a hernia and is always identified directly over the exit of the canal. The fat is seized with a pair of thumb forceps and is separated from the femoral vein and adjacent structures by careful blunt dissection.

When the sac is identified beneath the pad of fat, it is dissected free, opened between two clamps and then ligated as high up as possible.

Beginning at the medial side, the fascial suture is anchored into Poupart's ligament, is then passed across the femoral ring to be then anchored in the pectineal fascia. This suture is passed backward and forward until the hernial opening is entirely closed.

When the sutures are drawn sufficiently taut to make them lie flat, there will be no constriction of the vessels as they pass into the thigh.

The living fascial suture method of closure is of special value in recurrent femoral hernia and in femoral hernias of large size.

VENTRAL HERNIA

This type of hernia is usually due to wound infection but may be caused by prolonged drainage or faulty closure. In these cases the usual method of closure depends on the overlapping of muscle and fascia over the defect. As a result, a suture is usually placed under considerable tension and in larger defects it may be impossible to approximate the edges of the opening.

Living fascial sutures are particularly valuable in the cure of these cases.

Technique. Make an elliptical incision, excising the old operative scar. Deepen the incision very carefully, exposing the sac. The sac is opened with the greatest care since frequently the intestines will be found densely adherent to it.

When this condition is found to exist the intestines should be freed with a knife, leaving islands of sac adherent. Free the peritoneum from the overlying structures for about an inch on either side. Excise the sac and close with a No. 2 plain catgut suture.

The closure of the defect is effected with fascial sutures entirely. When the fascial edges of the abdominal wound can be approximated without tension this is done with two layers of continuous living fascial sutures.

In cases with larger defects no attempt is made to approximate the edges of the fascial wound, the defect being closed by weaving living fascial sutures across it, as one would darn a hole in a sock.

The author has personally performed thirty-five fascial repairs up to July, 1932.

Recurrent inguinal hernia	11
Direct inguinal hernia	15
Indirect inguinal hernia	4
Large ventral hernia	3
Femoral hernia	2
Total	35

CLOSING VERY LARGE VENTRAL HERNIAS

The repair of large ventral hernias with autogenous fascial sutures made from fascia lata has been quite satisfactory and there is no question that it has greatly reduced the incidence of recurrence and widened the field in which operation is indicated. There are some cases, however, in which the hernial ring is so large that no method of suture can adequately close the opening. There are others in which, while it is possible to draw the edges of the opening together by tight lacing of the suture, this can be done only with such an increase of the intra-abdominal pressure as to seriously endanger the life of the patient. In most cases the use of fascial sutures has proved successful but unfortunately, in a certain number, recurrences have developed which subsequent operation has shown to be due to protrusion through chinks between the sutures.

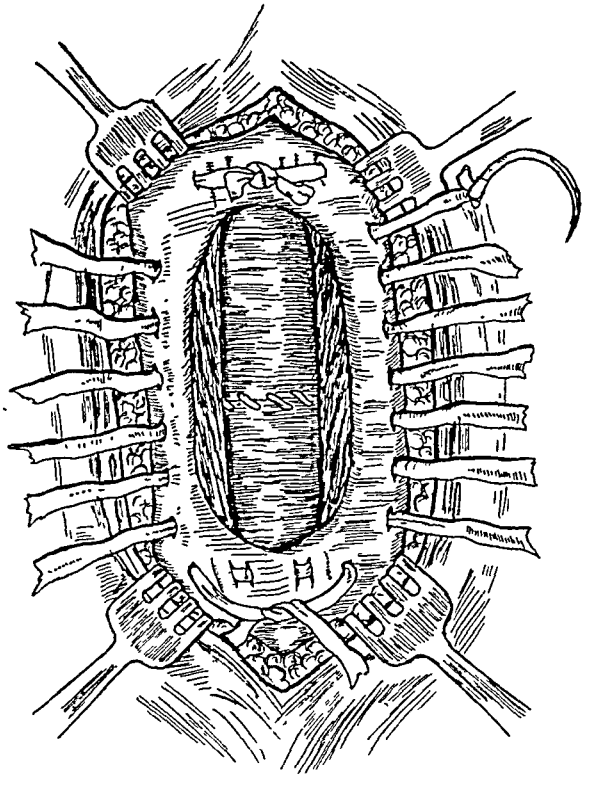


Fig. 8. Very large ventral hernia, opening closed by Gallie method. Two sheets of fascia lata are placed edge to edge and sutured; tails pass through edge of hernia ring and strips of one side of opening are tied to those of the other, bringing edges as closely together as seems desirable.

This has been found in a combination of the patch transplant with the living suture. It will be remembered that the defect in the patch transplant was that it could not be depended upon to heal strongly into the opening. There was no doubt that it could be expected to live but there was great doubt in regard to the strength of the scar and areolar tissue that joined its edges into the hernial ring. (Fig. 8.)

Technique. This uncertainty can be overcome by using as a patch transplant

a sheet of fascia lata whose ends have been split into quarter-inch strips so that it resembles the old-fashioned many-tailed abdominal bandage. When the strips have been threaded on needles they are passed through the thick margins of the ring and those of one side of the opening are tied to those of the other, bringing the edges as closely together as seems desirable. In this way a strong aponeurotic structure, free from chinks and other weak spots, is securely fastened into the defect and may be depended upon to close the opening permanently.

The method is particularly applicable to large ventral hernias and most frequently to hernias resulting from suppuration following operations on the gall bladder and stomach. It is useful also in cases of large direct inguinal hernias, particularly those in which the abdominal wall is feeble and in which several unsuccessful operations had previously been performed.

In the case of the ventral hernias a few special points in technique should be observed. In preparing the hernial opening for the closure no attempt is made to separate the various layers from one another as this definitely weakens the wall. The peritoneum is pushed back from the edge of the opening for half an inch or more so that the needle may be passed from within outward through the edge of the ring without entering the abdominal cavity. If the hernial opening is longer than 3½ inches it will be necessary to cut two patches from the fascia lata which will be laid side by side across the opening. As a rule, two patches of fascia, 5 inches by 3½ inches, can be obtained from the lateral aspect of a single thigh. The ends of the patches are then split into strips about ¼ inch wide and by means of a fascia needle passed through the edges of the ring as shown in Figure 8. If a second patch is needed it is laid edge to edge with the first and the two are sewn together with a fine strip of fascia lata threaded on a needle. At the ends of the opening, special precaution is taken to weave the tails into the edge of the ring so as to prevent a protrusion over the end of the fascial sheet. When all the tails have been drawn through the abdominal wall they are tied together, each to its fellow of the opposite side, and the edges of the opening drawn together as closely as seems safe. If they can be brought into contact without too much tension, so much the better, but, if they cannot, the surgeon may rest assured that the deep side of the defect in the abdominal wall has been permanently closed off. Often it is possible, where the deep portion of the opening cannot be drawn together, to close the anterior rectus sheath with catgut or a suture of fascia and this shuts off the sheets of fascia from view and seems to be an advantage. Care should be taken to oversew the knots in the tails of fascia with chromic catgut as these have a strong tendency to slip and untie when strain is put upon them.

CONCLUSIONS

1. Attention is called to the great value of fascial sutures in the cure of direct inguinal hernia, large oblique inguinal hernia in patients past middle age, ventral hernia and large femoral hernia.

2. Living fascial sutures remain in situ indefinitely and if securely anchored and properly placed will close any hernial defect permanently.

3. Large ventral hernia can be cured only by use of fascial sutures.

4. The disfiguring scar, postoperative disability and operative infection has been largely eliminated by use of a fascial stripper.

5. The fascial separator and threader devised by the author greatly facilitate the fascial stripping operation.

Note: The instruments referred to are made by George Tiemann and Company.

SPIVACK'S METHOD OF GASTROSTOMY

WITH A BRIEF CRITICAL REVIEW OF SOME OF THE PREVIOUS METHODS AND A REPORT OF 3 CASES

NELSON H. LOWRY, M.D., F.C.S., AND S. SORENSON, M.D.

CHICAGO, ILL.

GASTROSTOMY is one of the oldest operations in the history of gastric surgery. There are reasons to believe that it was already known in the earlier part of the seventeenth century.

To our knowledge there are between thirty and forty methods of gastrostomy recorded in medical literature. The reason for this high number lies in the fact that all these methods proved to be more or less unsatisfactory and in many instances cause of untold misery and suffering for the patient.

Ever since Egelberg, a Norwegian military surgeon, advised gastrostomy in cases of stricture of the esophagus, surgeons have been confronted with two main obstacles which have continued to be serious drawbacks to all the different methods of gastrostomy. Of primary importance is leakage of the gastric contents which occasionally, particularly in the pre-antiseptic days, results in fatal peritonitis but more often causes eczema of the skin around the gastrostomy opening which greatly interferes with the comfort and well-being of the patient.

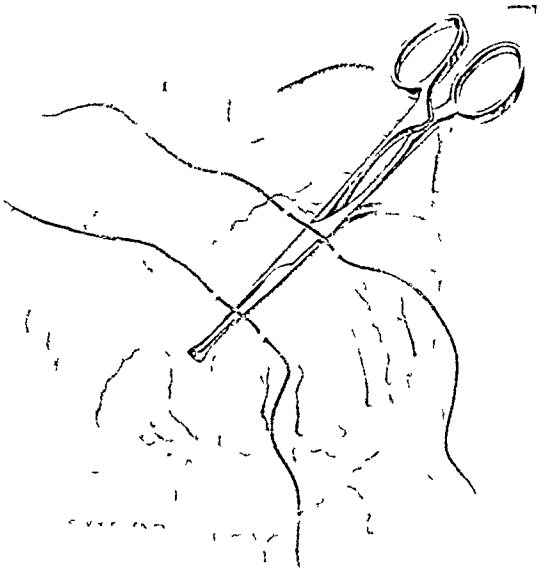


FIG. 1.

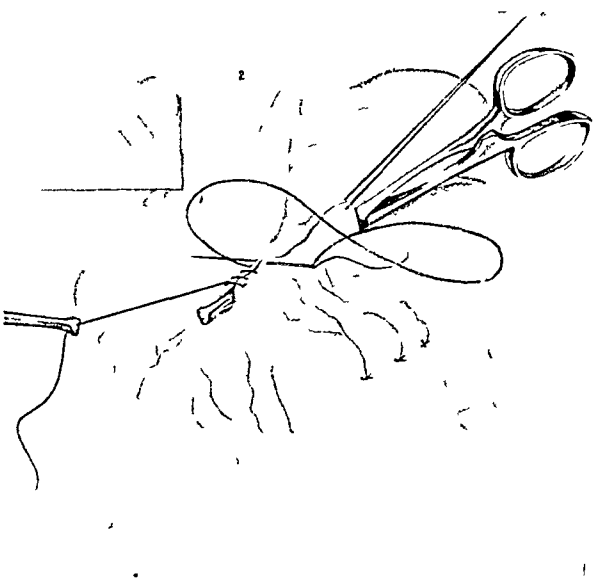


FIG. 2

Of secondary importance is spontaneous obliteration of the fistula which also occasionally happens, especially in cases where the fistula is lined with serosa. Quite frequently the obliteration can be so complete as to render feeding of the patient impossible.

It is not without some interest in this connection to observe the requirements for an ideal method of gastrostomy which would, of course, be an operation of choice and to see to what extent some of the older surgeons succeeded in obviating these two obstacles just mentioned.

An ideal method of gastrostomy should be:

(a) Absolutely "watertight," i.e. not permit leakage of the gastric contents which always is irritating and painful to the patient. This factor is of paramount importance and as is evident from the descriptions of all the previous methods, this leakage was the factor that surgeons attempted to overcome in different ways, sometimes paying meager attention to other factors.

(b) The canal should be lined with mucosa as the serosa has a tendency to obliterate when irritated by a foreign body.

(c) It should not require a large piece of the stomach as in most cases requiring gastrostomy the stomach is small and contracted.

(d) It should be simple in technique so as not to consume much time. With the introduction of blood transfusion and glucose solution intravenously or by proctoclysis and improved methods of anesthesia this factor is not as important today as it was fifteen or twenty years ago.

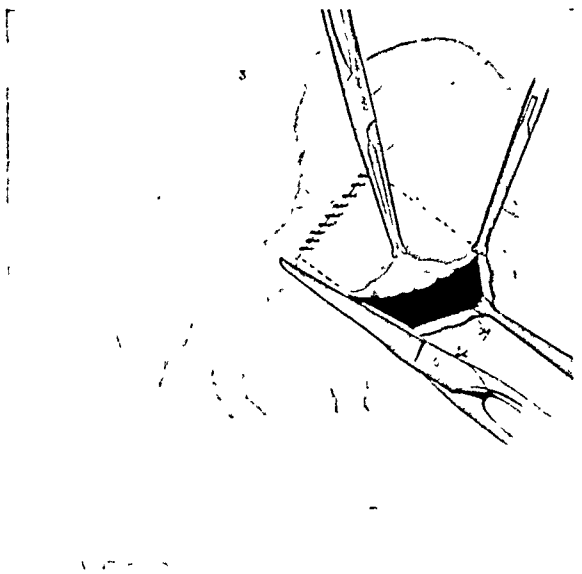


Fig. 3.

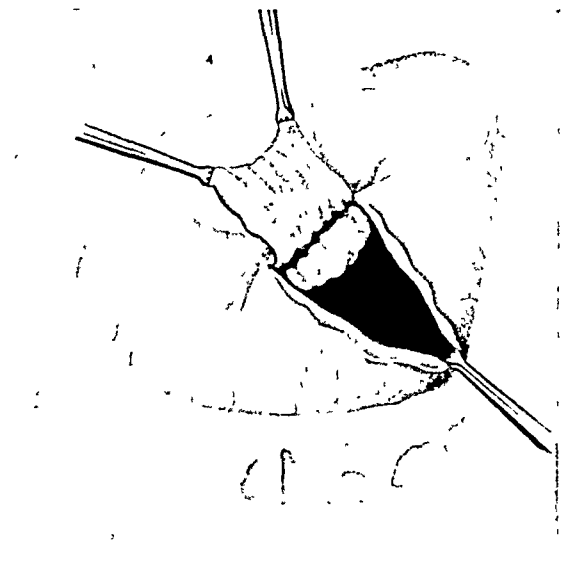


Fig. 4.

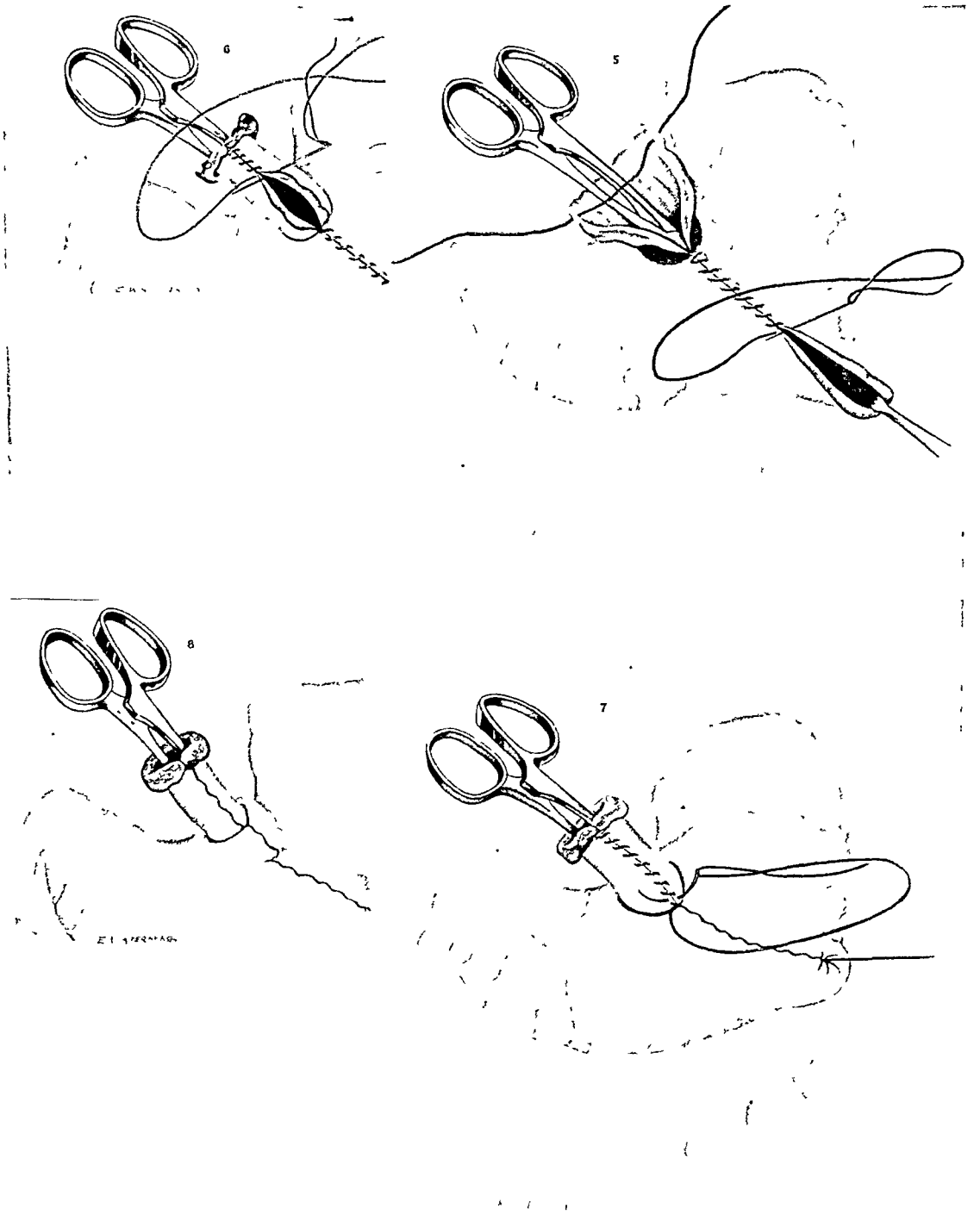
All these attempts to prevent obliteration of the canal and to make the fistula watertight may be classified and presented in several groups as follows:

1. A cone is formed from the anterior wall of the stomach so that the mouth of the cone is compressed by the surrounding structures. An example of this group is the method of *Hahn*. Hahn stitched the stomach in the eighth intercostal space in order to use the elastic costal cartilages as a compression stopcock which would prevent enlargement of the fistula and leakage of the gastric contents. Experience, however, has shown that the costal cartilages soon lose their elasticity and that this procedure is not proof against leakage.

2. Formation of a subcutaneous canal through which the stomach is pulled. Prevention of leakage is secured by applying bandages over the entire length of the canal. The methods of *Sabanejew-Frank, Jaboulay* and *Lucy* are examples of this group. Jaboulay and Lucy modified Sabanejew-Frank method and these modifications are said to have some advantages over the original method. However, this

method requires a large stomach and in cases where it is small and contracted this method would prove disadvantageous.

watertight results. An example is the method of *Ullmann* which does not need further description.



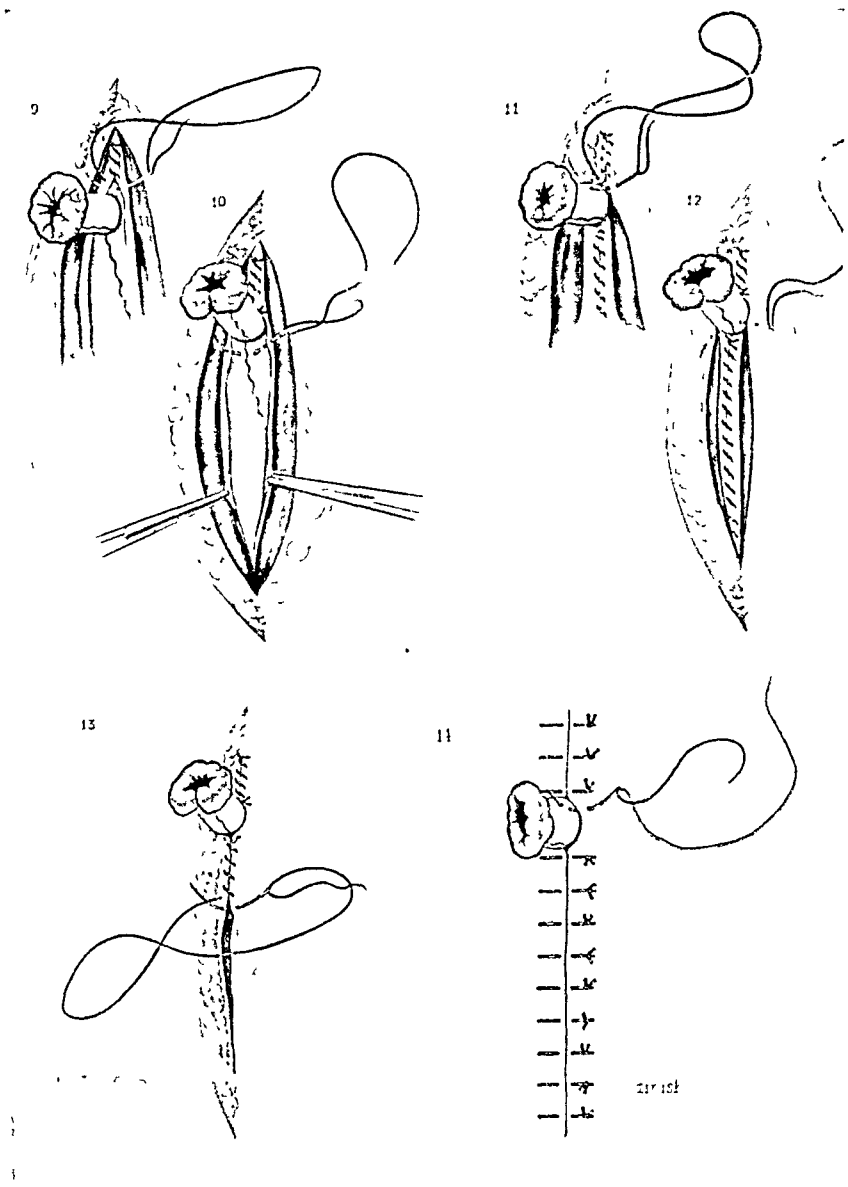
Figs. 5–8.

3. A cone is formed from the anterior stomach wall which is rotated 180 degrees at the external opening so as to secure

4. Formation of a sphincter from M. rectus abdominis. Examples are the methods of *Von Hacker* and *Girard*. Both

these methods are based on the idea that this artificial sphincter would be under the control of the will and that the patient would, therefore, have voluntary control of the gastrostomy opening. These methods never became practical on account of repeated failure to produce an artificial sphincter from a group of voluntary muscles.

5. Formation of an oblique canal in the anterior wall of the stomach. Example: method of *Witzel* and its modifications (Marwedel, Schnitzler). In this group can also be included *Stamm's, Kader's* and *Fontana's* methods. The difference between these methods and those of Witzel's type is that the canal runs perpendicular to the anterior wall of the stomach, whereas in Witzel's method it runs parallel. The disadvantage common to all the methods in this group is that the fistula is lined with serosa and tends to obliterate.



FIGS. 9–14.

6. Formation of a valve in a contracted cone from the stomach wall. Example: method of *Senn*. In fact there is actually no valve which would prevent leakage from the stomach.

7. Implantation of a loop of jejunum into the anterior wall of the stomach. An example is the method of *Tavel*. This is a very complicated procedure and the fistula formed is by no means watertight.

8. A small tube lined with mucosa is formed from the anterior stomach wall. Example: methods of *DePage* and *Janeway*. As the tube is lined with mucosa it does not obliterate but there is nothing to prevent escape of gastric contents.

In this classification we have included some of the best known methods of gastrostomy and examples of each group.

Spivack's method consists in making a tube from the anterior stomach wall *with a valve at the base*. The tube is lined with mucosa and does not tend to obliterate. Slightest intragastric pressure flips the valve into the internal opening of the tube and closes it so completely that not a single drop of gastric contents can escape.

As may be seen by comparing these groups, Spivack's method stands in a class by itself and is not a modification of any other method. It was first described by its originator in 1929.[1]

TECHNIQUE

The technique of this operation as described in the original article but somewhat modified by the originator is as follows:

Step 1. The stomach is brought out of the abdomen through a median or left paramedian incision. A quadrangular-shaped area 2 by 4 inches, depending, of course, on the size of the stomach, is mapped out on the anterior stomach wall. The upper half of this area is doubled according to the technique shown in Figure 1.

Step 2. A forceps is placed inside of the fold and the two ridges formed are united by a continuous suture (Fig. 2).

Step 3. The vessels at the greater curvature that are bound to be cut are carefully ligated. Two Allis forceps are then placed about 1½ to 2 inches below the ends of the fold previously made and at a right angle to each other. The stomach is cut open with sharp scissors along the greater curvature. The cut edge of the stomach is grasped with an Allis forceps right in the middle of the incision so as to prevent the stomach from slipping away and contaminating the field (Fig. 3). The opening is now extended toward the lesser curvature (Fig. 3) and a U-shaped flap made from the anterior stomach wall at the base of which is the fold previously made (Fig. 4). It should be borne in mind that the vertical incisions should *not* extend beyond the fold in order to secure watertight results.

Step 4. The opening in the stomach is sewed up with silk or tanned catgut sutures, starting at the base of the flap and extending toward the greater curvature (Fig. 5). In order to avoid catching the valve in the stitch, a forceps is placed inside of the stomach and the fold pushed away from the field (Fig. 5).

Step 5. The flap made from the anterior stomach wall is sewed up in a similar manner, starting at the upper angles, and a tube is made with a valve at the base (Figs. 5 and 6).

Step 6. The tip at the greater curvature is inverted by a purse-string suture and the whole suture-line is reinforced by a right-angle Cushing stitch. At the point of junction between the tube and the stomach wall, some kind of a cuff is made by taking a stitch in the tube just above the weak angle; thereby the tube is pushed a little downward so that the base of it is surrounded by the wall of the stomach (Figs. 7 and 8). This procedure prevents any possible leakage to take place from the angle and reinforces it considerably.

[1] *Bruns Beitr. z. klin. Chir.*, 147: May 10, 1929.

Step 7. The tube formed from the anterior stomach wall is now brought into the wound and the edges of the peritoneum are sewed to the stomach around the tube by two seromuscular stitches which are placed *at the base of the tube* but not in the tube itself (Figs. 9 and 10). This is an important step and should never be overlooked. The stomach, whether full or empty, has a tendency to pull the tube into the abdomen and any stretching or pulling of the tube during the first few days after the operation may result in necrosis of the tube or breaking of the cuff previously described and produce leakage from the angle with ensuing peritonitis.

Step 8. The fascia is sewed around the tube in a similar manner, two stitches being taken in the tube penetrating the seromuscular coats only (Figs. 11 and 12). After the wound has been closed up the skin edges are stitched to the top of the tube. The final results are shown in Fig ure 14. When doing these final steps, care should be taken not to constrict the tube.

According to the originator the principle involved in this method is based on the well-known fact that the surface of the mucosa of the stomach is much larger than that of the serosa and muscularis and as soon as the seromuscular coats are incised the mucosa tends to protrude through the incision. This fact is taken into consideration when making the valve previously described which closes the base of the tube hermetically and prevents leakage.

This operation is not technically complicated. In the hands of an experienced surgeon it should not take more than twenty minutes and it has been repeatedly shown that this method can be performed on a very small and contracted stomach. There is a case on record where it was performed on an infant two days old.

There are, to our knowledge, about 15 cases on record on which Spivack's method of gastrostomy has been successfully performed by different surgeons.

We take the opportunity of reporting here, briefly, the first 3 cases on which this method was performed, in order to show the advantages of this method and point out a few pitfalls in the technique.

Case 1. M. Z., white, male, seventy-eight years of age. Admitted to the Edgewater Hospital, Chicago, August 6, 1929. When admitted the patient stated that he had been well until about nine years ago. At that time he had swallowed a solution of some corrosive material which resulted in stricture of the esophagus and its characteristic symptoms. The patient was cured of this condition after six weeks of treatment and remained well until just recently. At the time of admission he was unable to swallow even liquids. *X*-ray examination revealed a large sacculated aneurysm of the arch of the aorta and a diagnosis of aneurysm of the aorta compressing the esophagus was made. Spivack's method of gastrostomy was performed by Dr. Julius L. Spivack and one of us (N.H.L.) under splanchnic and regional anesthesia as the patient was extremely dehydrated and a bad surgical risk. He survived the operation, however, but succumbed sixteen days later from lobar pneumonia.

No difficulty was ever experienced in inserting a rubber tube into the stomach for feeding purposes and no leakage was ever noticed. It is worth mentioning that at the time of operation the patient vomited when the gastric part of the operation was just completed. While straining the stomach became very much distended and the gastric contents were ejected through the constricted esophagus but not a single drop came out of the tube.

This incident demonstrated conclusively that the gastrostomy was absolutely watertight.

Case 11. G. M., white, male, forty-eight years of age. Admitted to the Edgewater Hospital, Chicago, August 16, 1929. Gradually over a period of one year the patient has noticed increasing dysphagia and lost about 20 lb. of weight. Difficulty was at first experienced in swallowing solid food but within the past few months the dysphagia has become so complete that at the time of admission the patient was unable to take food in any form. Consequently, the patient was very emaciated and dehydrated. The diagnosis was malignant stricture of the esophagus. Spivack's method

of gastrostomy was performed by Dr. J. L. Spivack and Dr. Loren Wilder, on August 17, 1929. The stomach was freely moveable but quite small and contracted.

The patient made uneventful recovery and was discharged from the hospital September 10 in an excellent condition. When last heard of, eighteen months after leaving the hospital, the patient was still alive and feeling quite comfortable. An interesting feature about this case was the fact that six months after the operation the patient came back to Dr. Wilder stating that some time after he went home he had discovered that he could swallow almost any kind of food without difficulty and that he had not been using the fistula for a few months. As he was again experiencing increasing difficulty in swallowing he came back to the doctor because he was afraid that the fistula might have closed up since he ceased using it. Even then Dr. Wilder found no difficulty whatsoever to introduce a rubber tube and feed the patient. It is worth notice that the patient never experienced any inconvenience or annoyance due to the operation. The skin around the opening was perfectly normal and showed no signs of irritation.

This case illustrates that the fistula is not only watertight but, also, that it has no tendency of obliteration even when left alone for several months.

Case III. M. A., white, female, aged fifty-two. Admitted to the Ravenswood Hospital, Chicago, October 2, 1929. The patient complained of pain at the lower end of the sternum of three weeks' duration which appears immediately after eating and is followed by nausea and vomiting. Lately she has been able to swallow liquids only. Has lost 30 lb. of weight in the last three months. *X*-ray examination of the esophagus showed barium remaining in the distal portion of the esophagus just proximal to the diaphragm. Diagnosis: Carcinoma of the distal end of the esophagus. Spivack's method of gastrostomy was performed by Dr. Loren Wilder. The stomach was free and moveable so that it could be brought forward. At the cardiac end was felt a mass about as large as normal kidney, hard and nodular and fixed under the diaphragm.

The patient made uneventful recovery and was discharged October 20 in an improved condition. She died fourteen months later from general carcinomatosis. Autopsy revealed that the whole stomach including the tube was involved but *the valve at the base of the tube was still intact and watertight.*

Another case of similar interest, operated upon by Dr. Spivack and Dr. Nelson M. Percy, is on record at the Augustana Hospital, Chicago. The tube sloughed away but even so there was no leakage from the fistula which showed that the valve was still intact. The patient died from bronchopneumonia on the seventh postoperative day. The stomach was found involved in a carcinomatous growth but was watertight.

The cases here reported and several other cases prove beyond all doubt that Spivack's method is superior to any other method of gastrostomy so far devised. The two major disadvantages common to most methods of gastrostomy, leakage and obliteration of the canal, have been completely obviated and as it fulfils all the other requirements of an ideal method of gastrostomy, it is, to our judgment, an operation of choice.

Provided the surgeon who attempts to perform this operation follows the technique here described, he can be assured of uniformly good results.

There is one point in technique we want to emphasize. *Never leave a rubber tube inside of the canal after completion of the operation.* This mistake may result in pressure necrosis of the tube made from the stomach wall and render feeding of the patient quite difficult. There are, namely, two cases on record where this occurred due to misunderstanding on the part of the interns. Fortunately the necrosis did not reach the valve so no leakage took place.

We wish to state, also, that Spivack's method is not only indicated in cases of permanent gastrostomy. One of us (N. H. L.) had some time ago occasion to perform this operation on a patient with a benign stricture of the esophagus so as to make it possible to dilate the esophagus from below. That was successfully done through

the gastrostomy opening. Few months after the patient was relieved and well the gastrostomy tube was removed and the stomach sewed up. The patient made rapid recovery. The feasibility of inserting radium seeds into the stomach in cases of carcinoma of the cardia may also be suggested.

SUMMARY

1. The disadvantages common to the previous methods of gastrostomy are: (1) leakage of the gastric contents, causing painful eczemas and (2) spontaneous obliteration of the fistula, rendering feeding impossible.

2. In order to overcome these difficulties a tube is made from the anterior wall of the stomach, lined with mucosa and does not, therefore, have a tendency to obliterate. At the base of the tube is formed a valve which prevents leakage.

3. This operation can be performed on a stomach of small size. It has been performed on an infant two days old (for tracheoesophageal fistula).

4. The technique is not complicated; however, the details should be meticulously followed in order to secure good results.

5. As this method fulfils all the requirements of an ideal method of gastrostomy it should be an operation of choice.

VAGINAL STERILIZATION

INDICATIONS AND OPERATIVE TECHNIQUE*

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CHICAGO, ILL.

STERILIZATION of the female is a recognized procedure and is performed as a prophylactic measure in order to save, or at least prolong, the life of the individual. The operation is done at the insistence of a specialist in one of the diagnostic fields of medicine because that physician feels if the patient were to become pregnant at a subsequent time her life might be endangered. The rationale of sterilization is to increase the span of life in those women who are afflicted with chronic disease which may become aggravated during the course of a pregnancy. This is true of all chronic diseases to which the human race falls prey with the exception of syphilis which is more amenable to treatment during pregnancy than it is at other times. If we continue to allow these women to conceive and carry on with their pregnancies we are taking unnecessary chances with the lives of our patients, we are adding unnecessary hours of worry and anxiety to our lives, and we are defeating all the other work we are doing in an effort to show a decrease in our maternal mortality.

The responsibilities of the medical profession lie in the direction of timely and adequate diagnosis of an existing medical, neurological, or mental condition. The surgeon should not take it upon himself to pass the severity of the existing condition or the amount of extra strain that that particular individual can stand in the form of pregnancy and labor. All patients in whom there is any small bit of doubt should have the advantage of a thorough examination by a competent specialist. It is only by such unselfish cooperation that the medical profession can honestly have all physically deserving patients sterilized, and by this means show much needed progress toward a substantial reduction in the maternal mortality statistics.

The commonly practiced contraceptive measures are, in a way, methods for temporary sterilization, but the uncertainty of their ultimate efficiency adds worry to the already troubled mind of the chronically ill patient. Our contention is that any contraceptive used over long periods of time is futile because it is either unsafe or adds further disease to that already present. Stem pessaries that are inserted into the cervical canal are actually harmful. Every patient who wears one of these devices develops an endocervicitis and cervicitis from the constant contact between the foreign body and the highly specialized cells of the endocervical canal. Leucorrhea always develops, and many women have a residual discharge that is so intractable as to necessitate amputation of the cervix or even total hysterectomy. In several cases which we have seen, the patients had presented themselves to their physicians for periodic removal and cleansing of the stem pessary. When the device was removed from the cervical canal virulent organisms were liberated, traveled through the pelvic lymphatics into the general circulation and fatal generalized sepsis resulted.

The cup pessary is undesirable for several reasons. It is necessary to remove the pessary before the onset of each menstrual period and to re-insert it after the period is ended. Thus it is necessary for the patient to visit her physician twice each month for removal and re-insertion of the cup. Many women do not have

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rigidly regular periods, and if a patient begins to menstruate at an unexpected time the cup blocks the cervical exit for the menstrual blood and there is a damming back of this blood. If the reflux is sufficient in amount it may spill through the fimbriated end of the tube and result in the very annoying and hard to treat endometriosis.

The use of medicated jellies or medicated douches is bound to fail in about 5 per cent of cases, and if we depend on them in our patients who must not become pregnant there is always the fear that the contraceptive will not prove successful. The semen is deposited in the posterior vault of the vagina, and quite frequently the patient fails to reach this place with the douche or the jelly and pregnancy may result.

The use of the condom is a comparatively safe measure. But even here unforeseen accidents occur and the purpose of the contraceptive is defeated. Not infrequently during the course of coitus the condom breaks and the parties concerned are unaware of this accident. Thus the relative safety of the condom is not absolute.

Finally, we have the commonly practiced coitus interruptus. If followed out properly this method is safe for the prevention of pregnancy, but the effects of this practice on the individuals using it may prove disastrous. Primarily there is the fear that premature ejaculation may take place and semen be deposited in the vagina. The nervous strain attached to this constant worry will eventually tell on the individual who has some illness which absolutely contraindicates pregnancy. Secondly, the persons who practice coitus interruptus withdraw before the orgasm is reached, and in this manner they are constantly being fomented to the point where pelvic congestion reaches a maximum without culmination of the act. This is an underlying factor in the production of so-called nervous individuals and is prone to produce irritable, dissatisfied, and nervously unstable patients. Thus we see that the practice of coitus interruptus adds nervous strain to an already damaged body and our medical task is made a more difficult one.

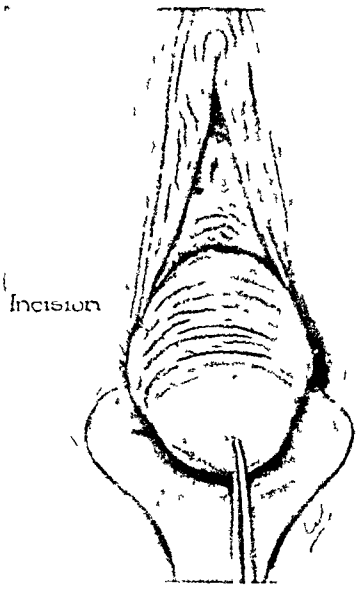


FIG. 1.

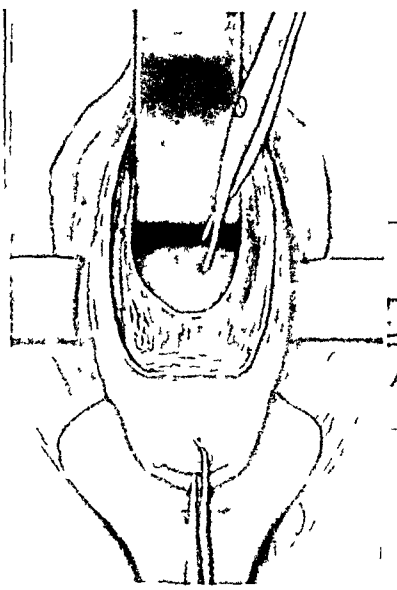


FIG. 2.



FIG. 3.

FIG. 1. Incision in cervical mucosa.
FIG. 2. Vaginal mucosa and bladder reflected. Peritoneum opened, uterine body visualized.
FIG. 3. A. Resection of tube and uterine cornu. B. Closure of uterine cornu.

After an analysis of the most common contraceptive practices we are forced to the conclusion that the patient who, for some medical reason, is in need of sterilization is not adequately protected by the use of contraceptives. We cannot rely upon any of the foregoing measures to keep our patients from becoming pregnant indefinitely. The obvious conclusion, then, is that actual sterilization by operation is the only procedure that will insure both sterility and the maintenance of the mental balance of the sensitive patient.

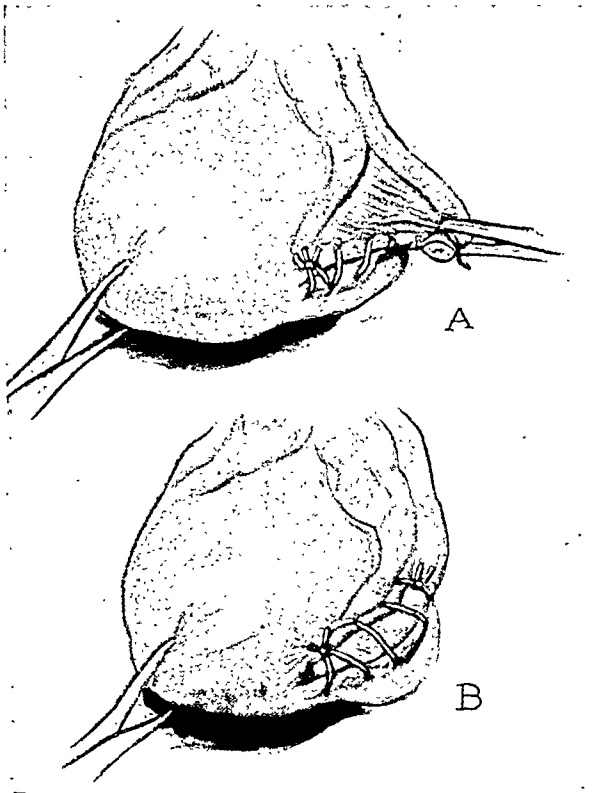


Fig. 4. A. Tube and broad ligament being brought over uterine horn. B. Peritonealization of uterine horn by tube and broad ligament. Note tube ligated by each stitch.

In performing the operation for sterilization we prefer to use the vaginal route and we advocate that route of attack to all surgeons who are called upon to do the operation because of its technical simplicity and because it is much easier on the patient. Gas anesthesia can be successfully employed for the vaginal operation, while the abdominal approach is difficult and at times even impossible if some ether is not used in order to get the proper amount of relaxation. If necessary local infiltration anesthesia may be used with little discomfort to the patient, but in doing laparotomy local anesthesia fails as often as it proves successful. So we see that from the point of view of anesthesia alone the ill patient is better off with the vaginal operation than when the abdominal route has been employed.

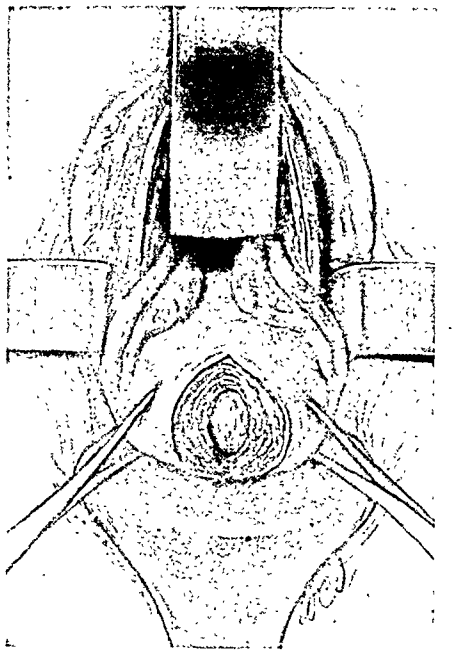


Fig. 5. Incision for fundal vaginal hysterotomy.

Any patient who has borne children has a sufficiently dilated vaginal tube to enable one to enter the abdomen from the vagina without difficulty. Most patients of this type require some vaginal plastic work which can be done in conjunction with the sterilization and thus save the necessity for a combined operation or for two separate operations. Even though the patient has not had children the procedure can be successfully carried out without a great amount of difficulty. If the vagina is not sufficiently relaxed a Schuchardt incision can be made and the vagina enlarged to enable of facile completion of the operation.

The vaginal route is also advantageous because it saves opening the abdominal wall and handling the intestines. This cuts down on the amount of postoperative discomfort, reduces to a minimum the amount of gas pains and intestinal distention, reduces the chances of a generalized peritonitis and ileus, and removes the possibility of mid-line abdominal hernia. Of secondary consideration are the time factors involved. The vaginal operation can be done in shorter time than an abdominal operation and thus the length of time of anesthesia is reduced. This may, in some conditions, be of distinct advantage to the patient. The vaginal route also proves to be an economic time saver because the patient is allowed out of bed and out of the hospital more quickly than she would be if laparotomy had been performed.

Finally, if therapeutic abortion is to be done at the time the patient is sterilized, both operations can be carried out at one time without any considerable increase in the operative difficulty, in the time necessary to complete the operation, or in the postoperative morbidity.

INDICATIONS FOR VAGINAL STERILIZATION

Cardiac Indications: Mitral lesions with myocardial changes are notably dangerous during pregnancy or labor. Any woman who has a cardiac lesion and in whom there has been at some previous time a cardiac decompensation should be sterilized. Such a patient runs a very great risk of developing another, and more severe, decompensation if she should become pregnant. Pregnancy is likely to terminate disastrously because of the extra load added to an already injured heart muscle. This applies to all previous cardiac decompensation whether occurring during the course of a pregnancy or not.

Mitral stenosis by itself does not constitute a hard and fast indication for sterilization, but any patient with a high grade mitral stenosis, with or without regurgitation, should be watched very carefully during the course of a pregnancy. If at any time in the first trimester she shows signs of cardiac embarrassment or beginning decompensation abortion should immediately be performed and she should be sterilized. Decompensations beginning in the second or third trimesters are best treated medically until viability of the child occurs, and then labor induced or cesarian section performed. This is advocated because labor or termination of pregnancy after the third or fourth month are as hard on the patient as full term delivery. Any woman who, after one or several pregnancies, develops a mitral valve lesion should be sterilized as soon as her medical condition permits. She already has her family and the danger of further pregnancies need not be hazarded.

Renal Indications: Every patient with a definite deficiency in kidney function should be sterilized because if a pregnancy ensues and the added work of elimination for both the mother and fetus is put on already damaged kidneys the result may prove dire. This is particularly true for patients who have had a true nephritis of pregnancy with marked edema, albuminuria, hyperpyrexia, and a retention of nitrogenous products as evidenced by the blood chemical findings. Unlike the acute toxemias of pregnancy, true nephritis does not entirely disappear after the termination of the pregnancy and the patients never return to their normal states. Therefore subsequent pregnancy is likely to prove of serious consequence.

Patients with tuberculosis of the urinary tract should also be sterilized because we know that pregnancy has an activating effect on tuberculosis and may stir up a quiescent affair. If the patient has had one kidney removed because of a tuberculous process she should be sterilized in view of the fact that most often renal tuberculosis is bilateral even though it may not show up in the opposite kidney for several years after the primary focus had been removed. If such a woman should

become pregnant a fatal outcome can be anticipated.

We do not want to convey the impression that every woman who has had a nephrectomy should be sterilized. We have delivered several patients who had had previous nephrectomy for calculi, and their pregnancies, labors, and postnatal courses did not deviate from the normal. Therefore we say that previous nephrectomy is not an indication for sterilization except in cases where the kidney had been removed because of tuberculosis or where the function of the remaining kidney is found, by laboratory procedures, to be below normal.

Pulmonary Indications: We all know that patients with active pulmonary tuberculosis should not become pregnant because of the activating effect of the pregnancy on the tuberculosis. The course of the tuberculosis becomes more rapid during the pregnancy. This does not mean that every woman who has had an active pulmonary tuberculosis should be sterilized. If a woman has had an arrested tuberculosis for five years with no evidence of activation during that time she may safely become pregnant and, under very careful supervision, carry to term with no ill effects. But if a woman with active tuberculosis periodically becomes pregnant and undergoes frequent therapeutic abortion, that woman should be sterilized. Or should a multi-gravida develop a pulmonary tuberculosis and progress is noted in the course of treatment of that disease she should be sterilized in order that a pregnancy should not interfere with the ultimate cure.

Otolaryngological Indications: One ear condition is known to be aggravated by pregnancy, many patients having no signs of this disease until they become pregnant. That condition is otosclerosis. If a patient suffering from otosclerosis becomes pregnant and the effect of the pregnancy on the ear condition is noticeably bad, that patient should have a therapeutic abortion and sterilization. If a patient with this disease presents herself with a history of previous gestation having caused a marked increase in the symptomatology, that patient should be sterilized because subsequent pregnancy will undoubtedly cause an increase in her difficulty especially since otosclerosis is a progressive disease. A nulligravida with otosclerosis and a great desire for a child may be allowed to become pregnant, but she should be warned that her ear condition will probably become worse. If the symptoms become so marked as to force the patient to seek relief she should be aborted and sterilized.

Tuberculosis of the larynx usually accompanies an active pulmonary tuberculosis, but rarely it may occur independently. If a patient has a laryngeal tuberculosis that patient should be sterilized under the same conditions and for the same reasons that a patient with active pulmonary lesions should have the operation performed.

Local Anatomical Indications: Local anatomical conditions, usually the result of pregnancy, frequently demand plastic repair, but these repairs are of no avail if the patient subsequently becomes pregnant and during the labor and parturition the operative repair is torn down. Large cystoceles and rectoceles, third degree lacerations of the perineum, vesicovaginal fistulae, rectovaginal fistulae, cervical lacerations extending beyond the internal os, and prolapse of the uterus fall in this group. All women presenting themselves for repair of the afore-mentioned conditions should be sterilized as a part of the operation in order to preserve the work done at the time of the operation. In doing the interposition operation the sterilization is an important part of the procedure, inasmuch as should pregnancy follow great difficulty will befall the patient and therapeutic abortion is frequently necessary to relieve a uterine or bladder incarceration. The only exception of this rule is the nulligravida with a uterine prolapse. This may be repaired without

sterilization in order to give the patient a chance to bear children.

Social Indications: Frequently we are approached by the social service departments of our hospitals and requested to sterilize certain patients for social reasons. Some of these requests we are forced to deny, but we feel that there are certain definite social indications for this operation.

There are apparently normal parents who have had several mentally deficient children. If this condition recurs in several children of the same parents there is a definite reason for sterilization in order to save these parents from bringing into being any more of those pathetic creatures who will eventually become wards of the state.

Another grave social problem is that of the unmarried mentally deficient girl who is promiscuous in her sex relationships and who becomes pregnant at frequent intervals. Sterilization of such an individual, with the permission of the parents and the juvenile court, is a desirable thing both from the standpoint of the family of such a girl and for the welfare of the community.

Amaurotic family idiocy is a rare condition that is prone to recur in certain families. If this condition is found in the offspring of one woman more than one time, that patient is entitled to sterilization in order to protect her from the danger of bringing more such infants into the world.

Neuro-psychiatric Indications: In some states there is a law making sterilization of the insane compulsory. It is definitely known that certain types of insanity have a tendency to recur in the offspring of the person afflicted. Many of these patients have recessions of their mental conditions, but if during such a period of betterment of condition that woman becomes pregnant there is likely to be a relapse of great severity. Therefore sterilization of such a patient is desirable both because of the good it may do the patient and because of the reduction in the numbers of the potentially insane.

Certain women, otherwise absolutely sane, develop a true psychosis during the course of a pregnancy. These patients have an absolute clearing of symptoms after the termination of the first pregnancy in which the condition developed, but with subsequent pregnancy the mental upset becomes worse and of longer duration. Such a patient should not be allowed to go through more than one pregnancy because of the danger of a permanent return of the previously encountered mental condition. We also see cases of puerperal psychosis in patients who go through their pregnancies and labors normally but during the course of the puerperium develop a psychosis. The majority of these women recover eventually, but to allow such a patient to again become pregnant is not good judgment inasmuch as she may develop a more severe and more permanent psychosis during her next puerperal period. Sterilization of women in this group is strongly advocated.

Chorea gravidarum, a very severe neurological condition that occurs in the course of pregnancy, is very likely to again appear during the next period of gestation in a more severe form. The choreiform movements may become so violent that the patient cannot control herself sufficiently to maintain her equilibrium. Falls are very frequent during these attacks and at times there are such severe injuries resulting from these falls that the patient is permanently crippled or dies. There are also cases reported where the movements were so violent and of such constant character as to completely exhaust the system and end in death from cardiac failure. Inasmuch as this condition is likely to recur during subsequent pregnancies, it is safest to sterilize the patient after the termination of the first pregnancy in which the chorea was observed.

True epilepsy is another indication for sterilization both because the patient herself is endangered and because the disease may be transmitted to the offspring. The danger of the patient lies

in the direction of increasing the severity of the epilepsy. It is known that the siezures are of more frequent occurrence and of greater severity during the course of a gestation, and following multiple pregnancies there may be a permanent change for the worse in the condition of the individual. Although the transmission of epilepsy to the offspring may be debatable there can be no question that if a mother with this condition has a child who shows an epileptic tendency, that woman should be sterilized in order to prevent her bringing more of her kind into being.

The rare neuromuscular conditions, pseudo-hypertrophic muscular dystrophy, progressive muscular atrophy, and myasthenia gravis are all characterized by a muscular weakness. If a patient with any one of these diseases becomes pregnant and extra weight is added to that which the weakened muscles must support, the general condition of that patient will suffer and the muscular condition will become aggravated because of the added strain. If patients suffering from these conditions are sterilized and the possibility of pregnancy is removed, the treatment of the neuromuscular disease can proceed without interruption.

THERAPEUTIC ABORTION

Unfortunately, many of the patients suffering from these conditions do not present themselves for examination until after pregnancy has taken place. Our problem now is twofold, doing away with the pregnancy and sterilizing the patient in order to obviate the possibility of future pregnancy. But even this double problem need not be difficult. In employing the vaginal route of operation the therapeutic abortion can be done as a part of the sterilization operation without adding much to the technical difficulties of the procedure. In doing the combined sterilization and abortion we prefer to do a fundal vaginal hysterotomy instead of the traditional transcervical uterine evacuation. In doing the operation by this method we do not go through the frequently infected cervix and so cut down on the chance for postoperative infection. In following out this technique we do not encounter sepsis such as we see on occasion in the patients who have had pregnancy terminated by the transcervical approach.

OPERATIVE TECHNIQUE

Sterilization can always be accomplished by the use of radium or the *x*-ray, but we rarely employ these methods of procedure because of the disadvantages attendant on their use. The *x*-ray and radium sterilize by a destruction of the ovarian function. This means that the patient will be thrown into the menopause, and menstruation will cease. In many patients the cessation of menses will prove a damaging psychic factor, while in others the symptoms accompanying the menopause may be very severe. Therefore we advocate the use of these forms of sterilization only in women who are approaching the age of the menopause or in those who have such serious medical conditions as to make even the very simple vaginal operation dangerous.

It must be remembered that in the course of the operation the abdomen is opened through the vagina and we must be very particular about the preoperative preparation of these patients. The external genitals and vaginal tube should be thoroughly washed with tincture of green soap and sterile water three times, with great care to get up to and behind the cervix. The soap is washed out with sterile water or with 1:5000 bichloride of mercury solution. The vagina is then thoroughly saturated with one-half strength tincture of iodine or the surgical preparation of mercurochrome, special attention being given to the posterior vaginal vault and the cervix. When the cervix is brought into view it is again swabbed with the iodine. These simple precautions will go a long way toward reducing to an absolute

minimum the chances of postoperative infection.

We will describe the technique that we use for simple vaginal sterilization, that for the repair of cystocele in conjunction with the sterilization, and that for the performance of therapeutic abortion during the course of the sterilization operation.

Simple Sterilization: The perineum is held back by the use of a weighted vaginal retractor. The anterior and posterior lips of the cervix are grasped by single tooth volsella tenaculae, and the uterus is pulled down into the vagina. Two cubic centimeters of obstetrical pituitrin are injected into the cervical tissues in order to promote uterine contraction as well as generalized contraction of the pelvic vessels and so cut down on the amount of bleeding encountered during the course of the operation. An incision is made across the anterior lip of the cervix (Fig. 1) just below the insertion of the bladder on the uterine wall. This incision is carried through the vaginal mucosa and submucous fascias. These fascias are then separated from the uterine wall, from side to side and upward by means of sharp dissection. A plane of cleavage will soon be found which will enable one to strip the bladder and its attached mucosa from the anterior wall of the uterus by gentle pressure with the gauze-covered finger. This separation of the bladder from the uterus is carried up to the point where the peritoneal reflection is brought into view. A narrow anterior retractor is now inserted between the reflected bladder and the uterine wall, and traction on this retractor gives one a good view of the peritoneum. The peritoneum is now incised and this incision is widened by inserting both index fingers and pulling them apart. This brings the fundus of the uterus into view and the anterior retractor is now placed between the upper peritoneal flap and the uterus (Fig. 2). The cervix is now pushed backward into the posterior vault of the vagina and in this manner the fundus is forced down toward the vaginal opening. The uterus is now grasped with a single toothed volsellum forceps, and by gentle traction is delivered into the vagina. This brings into view the entire uterine body together with the tubes and their insertions in the cornua.

The tube is grasped with a hemostatic forceps about ½ inch from the uterine horn and cut across. This incision is carried parallel to the tube as far as the uterine horn and across this, removing with it the interstitial portion of the tube (Fig. 3A). The uterine horn is now closed by a continuous running suture of chromicized catgut interlocked every second or third stitch (Fig. 3B). Care must be taken to bury the cut end of the interstitial portion of the tube. The proximal end of the cut tube is then brought over this portion of the uterine horn and sutured. In sewing the tube and its broad ligament attachment to the uterus care is taken to go around instead of through the tube. Thus we are peritonizing the raw edges of the uterine cornua and at the same time each stitch is surrounding and constricting the tube so that maintenance or reestablishment of the patent lumen is impossible. The same procedure is carried out on the other side (Fig. 4B). In the large series of cases in which we have employed this operation we are not aware of a single instance in which it has failed to sterilize permanently.

The uterus is now replaced in the abdominal cavity and the anterior retractor is taken out from beneath the peritoneal flap and reinserted beneath the bladder. The index finger is put into the abdominal cavity and the peritoneal edge is found and secured by a suture. The peritoneum is closed by several interrupted sutures of catgut. The anterior retractor is now removed and the bladder together with the attached mucosa is pulled down into place. The vaginal mucosa is now closed by interrupted sutures of chromic catgut.

Sterilization with Repair of Cystocele: If cystocele is present and operative treatment is decided upon in conjunction

with the sterilization, the technique is somewhat varied. In these cases it is necessary to separate the bladder from the vaginal mucosa at the outset and then to separate the bladder from the uterine wall. The same primary incision is made through the vaginal mucosa and then a curved rounded tip scissors is insinuated between the vaginal mucosa and the bladder. After pushing the scissors as high as possible it is opened and in this manner the bladder is separated from the vaginal mucosa. The mucosa is now split up the mid line as far as the separation has been completed, and in repeating the process the division of layers is carried on as high as the urethra. Lateral separation of the mucosal flaps is very readily carried on by gentle pressure with the gauze-covered finger.

The bladder is now separated from the anterior uterine wall as previously described, and the sterilization operation is carried out. After the sterilization is completed and the peritoneum is closed we proceed with the repair of the cystocele.

The vaginal mucosa is trimmed in order to tighten the covering of the bladder when the repair is completed. The lower edge of the bladder fascia is now caught and sutured to the uterine wall 1½ to 2 inches higher than its previous insertion. This elevates the bladder on the uterus and prevents it from coming lower down on the cervix. The bladder is sutured to the uterus with three interrupted sutures, one in the mid line and one on either side. The subpubic fascias are now brought together from side to side. This tightens the urethra and acts as a support for both it and the bladder. The vaginal mucosa is now brought together in the mid line by a continuous interlocked suture of chromic catgut starting near the urethra and proceeding downward. When a point below the newly made insertion of the bladder is reached, the suture is brought through one side of the vaginal mucosa, through the uterine musculature, and then through the other side of the vaginal mucosa. This type of stitch is carried on down to the lower edge of the vaginal incision. By taking these bites through the uterine wall we form another barrier in the path of the bladder should there be a tendency for it to slide down, preventing such a possibility and at the same time eliminating any "dead space" that may exist between the wall of the uterus and the vaginal mucosa. The transverse incision across the cervix is closed by several interrupted chromic catgut sutures. A retention catheter is put in place and left for five to seven days in order that the bladder may be kept empty during this time. This emptiness of the bladder allows it to regain its muscular tone and shrink in size. Thus, with increased bladder tonus, there will not be a tendency for over-distention and the primary causative factor in cystocele is removed.

Sterilization with Therapeutic Abortion: If therapeutic abortion is to be performed in conjunction with the sterilization the technique is not varied until the uterus is delivered into the vagina. A transverse incision is made across the fundus of the uterus anteroposteriorly (Fig. 5). The incision can be made from side to side and carried on to the removal of the uterine cornua, but we prefer the anteroposterior incision because in the mid line of the uterus no major vessels or large branches of these vessels need be cut across and the operation is practically bloodless. When the uterine cavity is opened into the products of conception can be squeezed through the incision in toto. The decidua is lightly curetted out through the incision and all is very carefully sponged away in order to prevent the possible development of an endometriosis. The incision in the uterus is closed by a running suture of chromic catgut, and this in turn is covered by a serosal stitch of fine plain catgut for purposes of peritonization. The sterilization operation is now carried out and completed as described.

[For Conclusions see p. 539.]

RUPTURE OF A CORPUS LUTEUM

WITH INTRA-ABDOMINAL HEMORRHAGE SUBSEQUENT TO ACUTE APPENDICITIS

R. B. McKNIGHT, M.D., F.A.C.S.

CHARLOTTE, N. C.

RUPTURE of a graafian follicle or corpus luteum cyst with severe hemorrhage occurs infrequently enough to warrant report. From the scattered single case reports, it seems probable that this condition occurs more frequently than is generally supposed. There is evidence that in some such cases bleeding has ceased spontaneously. The literature has been recently reviewed by Johnson[4] and by Greenhill[3] and discussions of the physiology, pathology, symptoms, diagnosis and treatment have been presented.

Recently a patient came under my care who presented the bedside picture, first, of an acute appendicitis, and, later, that of a ruptured right ectopic pregnancy:

A white matron, twenty-eight years old, was admitted to the Mercy Hospital shortly after midnight, January 25, complaining of severe lower abdominal pain, marked shoulder-top pain, collapse and shock. The menstruation was always regular and normal. At the birth of her child, seven years previously, labor was difficult. There had been no other pregnancies.

The morning before admission she went to her doctor complaining of moderate generalized abdominal pain of several hours' duration. He found some tenderness and rigidity over the right lower abdominal quadrant, and, on pelvic examination, a cyst of the right ovary the size of a walnut. The patient was advised to go home and to bed; symptomatic treatment was prescribed and a policy of watchful waiting assumed. Later in the evening her pain increased and localized in the right lower abdominal quadrant and nausea and vomiting came on. He then made a diagnosis of acute appendicitis and advised surgical relief. As she got into her car she experienced a sudden stabbing pain in the lower right abdomen, this in addition to the pain for which she sought relief. During the 25 mile ride to Charlotte, she complained of about as much right shoulder-top pain as abdominal. On admission her doctor said he thought she was suffering from an acute appendicitis, that there was an ovarian cyst present, and if the abdomen were opened he wanted the gall-bladder examined on account of the shoulder pain.

The patient was acutely ill. Blood pressure was 100/68, pulse 114, weak and of poor volume, temperature 99.4°F., respirations 24. There was an anxious expression on her face. The chest was entirely negative. The abdomen was tender and painful throughout, especially in the right lower quadrant, where there was also definite rigidity. A distinct fluid wave could be elicited. There was fluctuation in both fornices with marked tenderness on the right. The urine contained a trace of albumin and an occasional pus and blood cell; hemoglobin was 80 per cent, leucocytes 14,600.

We then made a diagnosis of ruptured ectopic pregnancy and, under nitrous oxide-ether anesthesia, opened the abdomen through a right rectus incision. On exposing the peritoneum, blood was seen underneath. The abdominal cavity contained 500 c.c. of fresh blood which was removed by suction, but kept accumulating. A quick examination revealed normal tubes and a typical acutely inflamed appendix. It was thought best to remove the appendix rapidly and then search for the source of the hemorrhage. The bleeding was found to come from a ruptured corpus luteum of the right ovary, in the depths of which was a small spurting artery. The area was resected by making a v-shaped incision and suturing in the usual manner. Sufficient peritoneal toilet was done and the abdomen closed without drainage. She stood the operation only fairly well and was not in good condition at its completion. After intravenous saline and glucose and supportive treatment, she rallied and blood transfusion was not necessary. Her convalescence was uneventful and she was dismissed from the hospital the thirteenth day after operation.

DISCUSSION

This case is of especial interest from the viewpoint of diagnosis. With definite evidence of bleeding and a distinct fluid wave, the diagnosis of acute appendicitis naturally gave way to that of ruptured ectopic pregnancy. Right shoulder pain makes one think of possible gall-bladder disease. However, the referred pain of biliary disease is to the right subscapular region, to the midline between the scapulae, or even to the left subscapular: it is not shoulder-top. Cope[2] believes that shoulder-top pain in the presence of lower abdominal symptoms establishes a diagnosis of ruptured graafian follicle or corpus luteum; perhaps the same symptoms occur in ruptured ectopic pregnancy.

Another feature of this case is one brought out by Brakeley and Farr.[1] In studying the cases reported, in all but one they observed rupture occurred during the week preceding the onset of the menstrual period. This was true of the case under discussion. Her period began three days after operation and she stated that it was her regular time.

Undoubtedly rupture occurred when she was getting into the car, as it was then she had the sudden stabbing pain and shortly afterwards pain was felt in the shoulder tops. Had the referring physician gone a step further he would have made a diagnosis which is seldom made, that of a ruptured corpus luteum with intra-abdominal hemorrhage, immediately preceded by an attack of acute appendicitis. The usual preoperative diagnosis in ruptured corpus luteum is either acute appendicitis or ruptured tubal gestation. In the words of the late John B. Deaver: "the aseptic scalpel dispelled doubt and revealed the truth—the whole truth!" Practically all of these cases will be diagnosed after the abdomen is opened; if we are alert we can make more correct diagnoses with the abdomen unopened. One can at least be sure that there is an intra-abdominal lesion demanding immediate surgical intervention.

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CONCLUSIONS OF DR. KANTER & DR. KLAWANS*

1. Sterilization of a woman is often necessary in order to save or to prolong the life of the patient.

2. Definite indications for sterilization exist, and the duty of the physician is to recognize these indications at the earliest possible time.

3. The use of contraceptives as a permanent method of sterilization is futile due to their frequent failure.

4. The vaginal route for the operative procedure has many distinct advantages over the abdominal approach.

5. Vaginal plastic operations and therapeutic abortion can be carried out as a part of the sterilization operation.

6. The indications for sterilization are given. They are divided into the following classes: cardiac, renal, pulmonary, oto-laryngological, local anatomical, social, and neuropsychiatric indications.

7. The fundal hysterotomy for therapeutic abortion is preferred over the transcervical route.

8. The operative techniques for vaginal sterilization, vaginal sterilization combined with cystocele repair, and vaginal sterilization with therapeutic abortion are described in detail.

*Continued from page 537.

A SIMPLE METHOD OF

SUPRAPUBIC CYSTOSTOMY

NORVELL BELT, M.D.

WASHINGTON, D. C.

NUMEROUS methods of opening a bladder by the suprapubic route have been devised with an attempt to speed technique on patients where time is especially important. The following is presented because of its simplicity.

The bladder is exposed in the usual manner and the site of puncture selected. An ordinary operating scalpel is introduced through one of the fenestra of the suprapubic catheter, penetrating the tip as far as the hilt. The mushroom is obliterated by traction on tube and bladder wall retracted with forceps. Knife blade with tube attached is inserted. The viscus must be thoroughly distended to prevent intravesical trauma. Withdrawal of the blade flattens the mushroom and the catheter remains within the bladder. Sutures are placed and closure completes the operation.

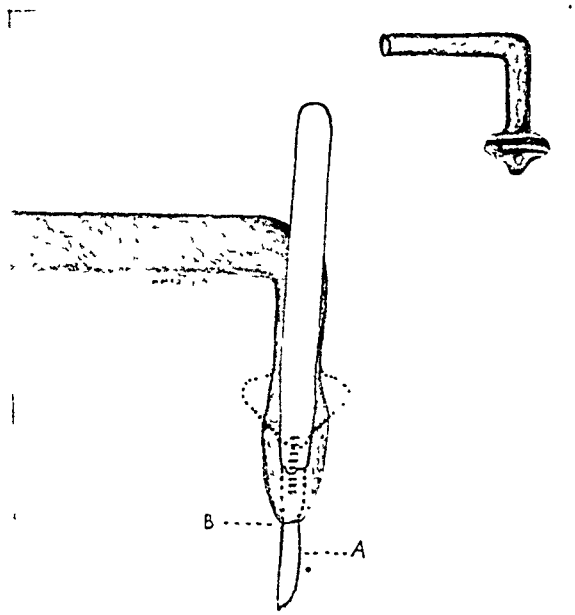


FIG. 1. A, Knife blade has pierced tip of catheter. B, Impinging hilt acts as obturator in obliterating mushroom. Inset shows ordinary mushroom catheter.

This procedure does away with special instruments and minimizes the flooding of the operative field which hampers closure. Shock is lessened by shortening of the operating time.

A COMFORTABLE AND SANITARY COLOSTOMY BAG*

E. R. ARN, M.D., F.A.C.S.

DAYTON, OHIO

THE need for a mechanically simple, comfortable and sanitary colostomy bag is apparent. Many of the colostomy outfits now available are complicated, provide a source of discomfort to the patient and are most difficult to keep clean. The purpose of this communication is to describe a colostomy outfit which overcomes all of these difficulties.

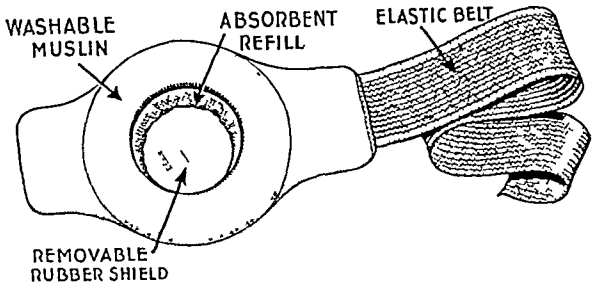


FIG. 1.

Mr. E. D. Smith, Plant Engineer of the National Cash Register Company, of Dayton, Ohio, was subjected to colostomy two years ago. Mr. Smith experimented with all of the available colostomy outfits. His dissatisfaction resulted in the invention of the colostomy outfit which bears his name and is now procurable through trade channels.

The Smith Colostomy Outfit contains no metal parts. The bag proper is made of washable muslin, on the inside of which is placed a removable sheet rubber shield. A removable cellucotton refill is placed inside the muslin ring. One or two small squares of gauze are then placed at the bottom of the bag in the space in the center of the absorbent refill. The bag is then held firmly in place by a wide elastic belt. If any part of the bag should become soiled the entire outfit may be readily washed. The patients who have used this outfit during the past two years are enthusiastic over its advantages.

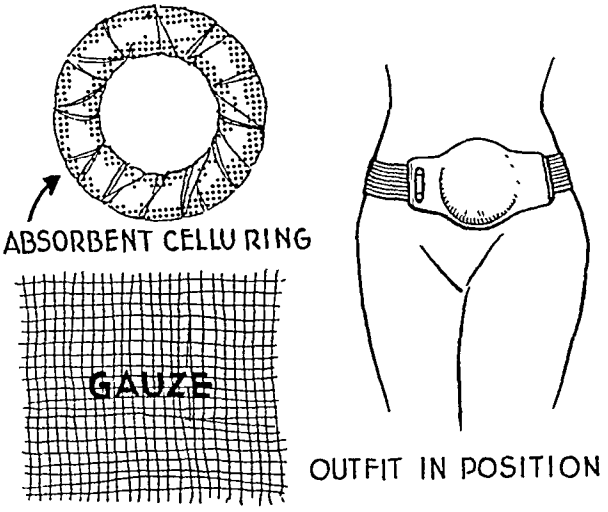


FIG. 2.

* From the Surgical Division of the Dayton Clinic, Dayton, Ohio.

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EDITORIALS

ASPHYXIAL DEATH—A PROFESSIONAL DISGRACE*

THE death rate from asphyxia is a reproach to the practitioner of medicine; first, because of figures available from municipal vital statistics; secondly, because the medical profession has practically turned over the treatment of asphyxia to a non-medical personnel.

The death-rate from asphyxia drawn from vital statistics of the City of New York for the year 1931 with a total registered population of 7,090,099 is more than twice the death-rate for auto accidents, 18 times the death-rate for diphtheria, and almost 40 times the death-rate reported for typhoid.

The argument may be advanced that we all die of asphyxia. The test of true asphyxial death, however, readily disposes of this contention. The generally accepted immediate treatment of asphyxia neonatorum, illuminating gas, submersion, anesthetic accidents, etc. is removal of the obstruction to the respiration and the application of artificial respiration. Prompt and efficient treatment along these lines usually results in cure. Failure to apply such treatment results in death from asphyxia.

In previous communications[1] the author has pointed out the fact that asphyxia is a generic condition, the specific instances of which are found in the various etiological factors listed below:

The segregation of various types of asphyxia, all subject to the same essential

[1] *Arch. Otolaryng.*, July, 1930; *Am. J. Obst. & Gynec.*, April, 1931; AM. J. SURG., 8: No. 6, 1930.

* A solution to the problem outlined above will be discussed in an editorial by Dr. Flagg in the next issue.

form of treatment, is the reason for the astonishing figure disclosed.

The following figures are on file in the Bureau of Vital Statistics, City of New York; for the year 1931, the total population then being reported as 7,090,086:

Submersion	442
Lightning	0
Electric shock	25
Drugs	0
Anaesthesia	42
Illuminating gas	305
Other gases	28
Mechanical suffocation	33
Conflagration	59
Total	954

It is fair to assume that the report of drug deaths and the report of anesthetic deaths are underestimated. Foreign body obstruction does not appear as such.

Of the total number of stillbirths reported (5579), the following may be classed as capable of response to resuscitation properly applied:

Cord pressure	535
Breach	140
Malposition	91
Deformed pelvis	91
Difficult labor	279
Asphyxia	282
Total	1418

If we reduce this figure by one third to allow for unavoidable deaths, we have a total of 946.

Antenatal and postnatal atelectasis results in asphyxial death, and may be treated and relieved, as is any other form of asphyxia, by intratracheal insufflation, under measured pressure.

The figure given for atelectasis (672) is not exclusive, as it includes icterus, sclerema, etc. For the sake of being conservative, we may reduce this figure one half, and accept a total of 336.

Pulmonary complications occurring during the first month may be regarded as due, in part, to aspiration pneumonia following birth, consequent upon a failure to remove fluid from the baby's pharynx and trachea. The figures for lobar pneumonia are 55; for bronchopneumonia, 181; allowing one third for pneumonia from other causes, we have a total of 158.

Premature infants dying in the first day frequently lose their lives from inefficient means of resuscitation. The total figure is 852. Allowing 50 per cent off of this figure, we have 426.

The totals of the various types of asphyxia are, therefore, as follows:

General asphyxia	934
Stillbirth .	946
Atelectasis	336
Pulmonary complication	158
Premature	426
Total	2800

Comparative figures from various causes of death are as follows:

Death from	
Typhoid	77
Diphtheria	156
Auto accidents	1298
Asphyxia	2800

If it be advanced that many asphyxial deaths, such as from submersion, lightning, conflagration, etc. are quite outside the possibility of rescue, it should also be recalled that many auto deaths are in the same category.

Why, it may be asked, was this situation not known long ago? The answer lies in the segregation of the specific instances of asphyxia into a single group, responding to a common treatment.

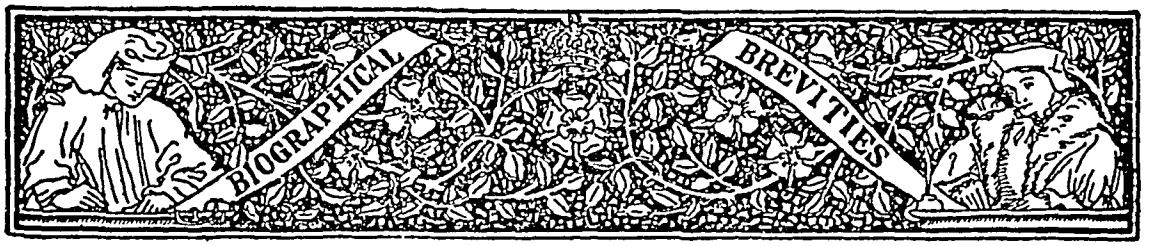
Why, it may be asked, has the Medical Profession been so remiss in its care of these patients? The reason is, that until within the last three years, no medical technique, or armamentarium suitable for the use of a physician was available.

The problem before us is a significant one from an economic point of view. Based upon a capital value of $5,000 for a life, we have a capital loss of $14,000,000. for New York City alone; or, or a quarter of a billion dollars for the United States with a population of 125,000,000.

Much of this life and much of this economic value may be saved by acknowledging the problem before the nation.

Approximately two-thirds of these lives are newborn; physically well when rescued. Would it not be worth every effort that we could make to preserve these to posterity?

Paluel J. Flagg, M.D.



AMERICAN PHYSICIANS

THOMAS CADWALADER

THOMAS CADWALADER of Philadelphia was born in 1708. Properly, he belongs in that group of medical giants who were responsible for Philadelphia becoming an important medical center.

While he was yet a youth (eighteen or nineteen years old) his father sent him abroad to complete his medical education. On his return to Philadelphia he soon won favor and acquired a remunerative practice. We are told that upon his return from Europe, Cadwalader "made dissections and demonstrations for the instruction of the elder Dr. Shippen and some others who had not been abroad." Wistar says the place these instructions were given "was in a building on the back part of a lot, on which the Bank of Pennsylvania now stands."

After his marriage (1738) to Hannah Lambert of New Jersey, he spent a great part of his time for several years in that state near Trenton, returning to Philadelphia about 1750.

With Franklin he was one of the founders of the Philadelphia Library. He was one of the American pioneers to adopt and practice the method of inoculation as a preventive against smallpox.

One of the high lights of his career is that he performed an autopsy in 1742. It is claimed to have been the first scientific autopsy performed in this country.

Cadwalader was one of the founders of the Pennsylvania Hospital. Also, he was a trustee of the Academy and College of Philadelphia. He was one of the charter members of the Philadelphia Medical Society. When the American Society for Promoting Useful Knowledge was consolidated with the American Philosophical Society (Franklin was President) in 1768, Cadwalader was the first named of three vice-presidents.

His services during the Revolution seem to have been those requested of him by Congress, and in assisting his friend, Dr. Morgan, at that time director-general of the military hospitals.

Only one publication bears his name: "An Essay on the West India Dry Gripes, to which is added an extraordinary case in physics. Philadelphia. Printed and sold by Benjamin Franklin, MDCCXLV." It is said to be one of the earliest medical monographs published in America.

Thomas Cadwalader died November 14, 1779, in Philadelphia.

T. S. W.



THOMAS CADWALADER

[1708-1779]

The American Journal of Surgery
N S Vol XVIII, December 1932



[From Fernelius' Universa Medicina, Geneva, 1679.]

BOOKSHELF BROWSING

BOUND FEET IN CHINA

F. M. Al-Akl, M.D.

NEW YORK

FOOTBINDING of Chinese females, a practice that fortunately seems to be rapidly disappearing from many of the Eastern provinces of China, is still rather prevalent in the country's interior, where the number of better educated females is too insignificant to exert any influence upon the communities in which they live, and where the inhabitants have hardly any contact with foreigners or foreign civilizations. Should China, however, keep up its present pace of progress, the custom in another century or so will inevitably become nothing more than one of the sad reminiscences of an agitated and tumultuous past.

The practice, which may possibly date much earlier, is believed to have been started by the Empress Taki about the year 1100 B.C. Having been born with club-feet, the Empress is said to have issued an order, on ascending the throne, exacting the binding of all female children's feet living in her domain, so they might thereby aquire an appearance analogous to that of their sovereign. Thus the style is supposed to have begun, and in time came to be handed over from mother to daughter as a fashionable practice. Again, some say that Emperor Gangti, completely fascinated by the graces of his most favorite concubine, had her feet bound. When their size was reduced to what seemed to him the most pleasing proportions, he presented her with a pair of very small shoes upon whose soles was carved a lotus flower to decorate her tiny footprints. It is, however, the belief of many Chinese that the custom was initiated and propagated by a desire among men to encumber their females in walking, in the hope of limiting their freedom and in coercing them to stay at home. But even at that, the men seem to have had little success, as in time women learn how to hobble about, without minding much the difficulties encountered in attempting to balance the body on their deformed extremities. Their gait, however, changes considerably, becoming peculiarly stiff and non-elastic, simulating one of a person who tries to walk on his heels over a wet floor.

The evils of this operation seem to have been realized time and again in China, and various attempts have been made to abolish this practice, without much avail. As early as 1664 the Emperor passed a law prohibiting footbinding, but the unsuccessful result occasioned by the lack of cooperation on the part of the subjects led, after a trial of four years, to its amendment.

The binding usually begins between the ages of four and eight, most often about six or seven, after the child is thor-



Fig. 1.

oughly well able to walk, and is continued throughout the entire life of the individual. No periods of rest or intermission, not even an overnight's relaxation is allowed and the victim is eventually placed in her coffin with the bandages tight on her cold feet. So when the time comes, the mother, especially if she is young and inexperienced in binding a youngster's feet, or if she is afraid lest her sympathy should betray her at the sight of her own child's tears, she calls to her aid an older and more experienced neighbor or possibly her customary midwife. In the case of the very rich, the bandage material is of silk, though usually strips of ordinary native cotton cloth or pieces of imported muslin are employed, each measuring about 3 to 5 feet in length and from 2 to 3 inches in width, depending largely upon the size of the foot. The number of strips is determined by the amount of tension to be exerted, but two to four usually suffice for each foot. The cloth is ordinarily plain, sometimes oiled. No preliminary preparation is required, and after the foot is kneaded a little, the cruel strips of cloth are tightly bandaged on. More vigorous kneading however is done after the bandages are applied. So severe are the manipulations that a fractured bone or the dislocation of a joint may occur.

The procedure consists in first diminishing the size of the foot by flexing the toes down upon the sole around its long axis. This is accomplished by the primary circular bandage started on the dorsum and wrapped around from the inner to the outer border. The great toe is the only toe



Fig. 2.

left in its position of normal extension, though it inevitably becomes compressed with the rest of the foot. After the binding, the girl is made to walk in order to help re-establish circulation and, with tears in her eyes, the child attempts to help support her body on the tender feet by holding on to the furniture about the room. The bandage is applied for the first few times by the experienced neighbor until the mother aquires the technique herself. The constantly reducing size of the foot tends in the beginning to loosen the bandages quite rapidly, consequently they have to be changed about once every day.

The toes being successfully flexed to a position beneath the sole, which operation may take a year, the second step in the process is more vigorously undertaken. This consists in diminishing the length of the foot by flexing it upon itself, thus approximating its front half to the heel. For the purpose, a figure of eight bandage is applied which compresses in its anterior loop the front of the foot and the heel in its posterior one, bringing them as closely together as possible. Thus, in addition to the actual interference with the normal

growth of the bone, the length of the foot is diminished by exaggerating the height of the plantar arch; the resulting fold in the



Fig. 3.

integument of the sole may be 2 to 3 inches in depth. Once the foot has been reduced to somewhere near the desired form, the various layers of bandage are secured together by a number of interrupted sutures. The bandage is then left for several weeks or months without being changed or reapplied unless it becomes loose or wears off; or unless the odor becomes exceptionally offensive, in which case a bath becomes unavoidable (a poor woman may not take a bath in the course of a whole year); or, unless the excruciating pain caused by the excoriation of the skin is rendered intolerable. By the age of eleven or so, the girl usually (acquires the art of applying) her own bandage as she begins to appreciate tiny feet and boasts of her own if they look any smaller than those of her companions.

The anatomical changes that take place in the foot are quite obvious and distinct. Viewed from above, the foot presents the appearance of a closed fist with a slightly hyperextended thumb. The projecting distal heads of the metatarsal bones resemble the knuckles of the fist when viewed from the same angle. The foot is very small and may be 2 inches at its broadest diameter. The nail of the great toe is almost cylindrical from lateral pressure. The contour of the dorsum is acutely angular. The tarsal bones curve sharply downward; the metatarsals run in an almost perpendicular plane. The sole is about triangular in shape



Fig. 4.

with the apex of the triangle at the tip of the great toe and the base towards the heel. Due to the weight sustained in walking, certain broad callosities are to a greater or lesser degree noticeable on what is now the plantar surface of the flexed toes. A deep fold divides the foot transversely and the skin of the sole looks tough and corrugated. The heel scarcely projects backwards and instead of the heel, a projection is usually noticeable over the posterior border of the ankle joint due to a backward displacement of the astragalus. Besides this forcing backwards of the astragalus, dissection reveals the peculiar position which the *os calcis* acquires. Instead of lying almost horizontally with its long axis running anteroposteriorly, the bone is found to have

rotated into a nearly vertical position whereby its long axis becomes almost continuous with that of the tibia. Its posterior

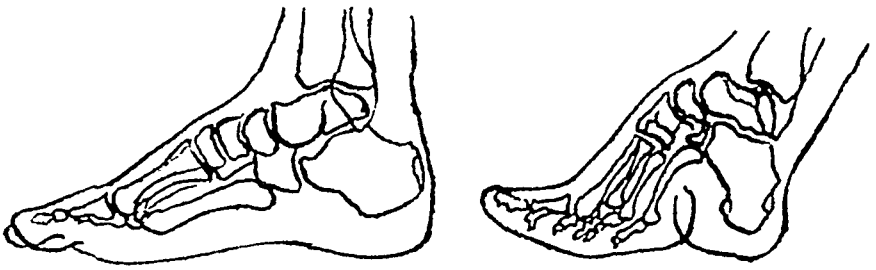


Fig. 5.

surface to which the tendo achillis is attached becomes the inferior surface on which the weight of the entire body is sustained. The other points of support on the plantar surface of the foot are usually the distal head of the first metatarsal bone and the once dorsal surface of the fourth and fifth phalanges.

The pain resulting from the manipulations is terrific and the Chinese proverb which says "each bound foot costs a tubfull of tears" seems to be no exaggeration. The mother may often, in the beginning, take a stick along with her to bed to beat the child to sleep should she wake up and cry, or the child may be left in some shed outside the house for the first few nights in order not to disturb the rest of the household. Later on in life, the unbound foot becomes the painful one for, as the bandage is removed, the foot looses its habitual support and the contracted structures give rise to a great deal of pain under the stretching-out influence of the weight of the body. For this as well as for other reasons, the binding is kept up all through life, more so because any negligence for even a few days may allow the foot to regain somewhat its normal contour and form. Some people use wooden mallets to help reduce the foot to the desired shape, some go, though rarely, as far as extirpating the scaphoid in an attempt to properly fold up and rigidly pack the foot. In some sections of the country, those who can afford to, use a semi-cylindrical piece of metal inserted transversely in the plantar fold and bind the foot firmly to it. With such measures, an adult foot may come to measure as little as 2½ inches from the tip of the great toe to the edge of the heel. Should excoriations of the skin develop, the foot may be bathed in plain warm water and a dry bandage applied. No particular medicine is used despite the variety of applications ranging from powdered tiger's bones to mortar from the Great Wall which native medicine men prescribe. This probably may be due to the fact that women are very much ashamed to talk about their feet, more so to show them to any one. As a matter of fact, they would consent to subject their genitals to an examining physician much more willingly than they would their feet; thus an excoriation may quite commonly develop into an ulceration and necrosis of the skin. The latter is more apt to occur in cases where the binding has been started rather late in life, apparently because the readjustments in circulation are so much more difficult then. The atrophy of the muscles of the calf is marked and the women, unable to balance themselves on the deformed feet, may often be seen kneeling down to perform the various exigencies which require the erect position.

Where footbinding exists, it is prevalent among all classes of people, more so among the well-to-do than among the poor. The Chinese consider small feet the outstanding feature of aristocracy and their largeness the extreme of vulgarity and plebeianism. The size of the foot, besides, seems to constitute the chief criterion of a woman's

commercial value. For when the woman is taken in marriage, her money is paid in inverse proportion to the size of the shoe she wears. If the shoes are small, the wooer usually has to settle the account in silver, if large the deal may be concluded in coppers with the loss of so much profit to the parents. It was this state of affairs that had for so long agitated the French sisters of Charity who, realizing the difficulties girls with large feet have to contend with in trying to find a suitable husband, consented most unwillingly to the footbinding of their orphans and boarding students.

The Chinese must obviously see certain beauty in small feet, hence their appreciation and predilection. Not only does the attraction seem to depend upon the mere smallness of the size, but also upon the consequences and after developments. The shortened pace and the slightly swaying coquettish gait seem to hold their special fascination and grace; so much so that in certain Tartar provinces where footbinding is prohibited, women use a peculiar type of footwear where the heel, instead of being at the back of the shoe, is placed about the middle in order that the Mongolian woman may be able to imitate her Chinese sister's gait by balancing herself on this centrally located heel.

Apart from this, there seems to be a strong sexual factor accountable in part for the long persistence of footbinding. The resulting atrophy of the muscles of the calf and the hypertrophy of the thighs, hips and mons veneris in particular is said to give the body a peculiarly exciting form. This probably is the reason why prostitutes attend to their feet with such fastidiousness and form the habit of bringing them together and sitting down on them when they want to rest, thus augmenting the pressure on their packed feet by the weight of their bodies.

Despite the many condemnations and all that may be said against the practice of footbinding, there unquestionably is a certain amount of taste in the underlying idea. The Chinese poet who wrote: "A large foot is a disgrace" must have had his reason; and despite the tendency to exaggerate the diminution of the foot's size, the notion may have been dictated by good esthetic sense. Even among Westerners, the tendency is to make the foot look as small as possible; and many a foot has been compressed and toes jammed together to the point of over-riding inside the diverse forms of narrow pointed shoes. Then again, should the theory so often quoted that man and the monkey come from a common origin be true, the Chinese would be assisting nature in diminishing the size of a foot no longer used in grasping branches or climbing up trees. Not only does the binding diminish the size of the foot, but by exaggerating the plantar arch and erecting the os calsis, it increases the length of the entire leg. The woman's waist is thus raised and her legs aquire a more artistic proportion to her trunk; Chinese women being certainly much more in need of that correction than white women.

The ill-effects of binding sustained by the foot as well as the system in general are innumerable. The legs which develop a variable degree of external rotation are spread apart in walking and the slightest mis-step may result in a severe fall. Thus walking and particularly running are greatly interfered with, the latter becoming practically impossible. Defecation is rendered very tedious and trying, for the stabilization of the body in the squatting position becomes exhausting if not impossible without some kind of support. The loss of the springiness of the normal foot exposes the spine to jarring if any amount of hurry is attempted. In addition to the excruciating pain, the various inevitable complications resulting from interference with the circulation are horrifying. Excoriations of the skin are to a greater or lesser degree present in every case. These may develop under the merciless persistence of binding into ulcerations which unavoidably become infected. The infection may travel down to the mutilated tissues and osteomyelitis may spread

rapidly in the devitalized bone once it has set in. The interference with circulation may be complete, in which case gangrene of one or more toes occurs. The gangrene may be dry or wet and may spread to the rest of the foot which not uncommonly drops off. One or more complications of sepsis in the foot may set in and end the victim's life. The ulcers may undergo malignant degeneration; suppurative inguinal adenitis, metastatic abscesses, erysipelas and septicemia may bring about a fatal issue. Should the individual however survive all these, the anemia and devitalization occasioned by lack of outdoor life, proper exercise, fresh air and sunshine in addition to the loss of appetite due to the obnoxious odors the feet emit, are most inviting to tuberculous infection which is always lingering somewhere in the filthy and unhygienic atmospheres of dwelling places and of which 10 per cent of the Chinese poor die yearly.

I wish to express my appreciation to Dr. James H. Ingram of Peking, China, who has practiced for forty-five years in China, for consultation in the preparation of this paper, and to Lewis Thorne for the illustrations.

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BOOK REVIEWS

MEN AGAINST DEATH. By Paul de Kruif, M.D. Harcourt, Brace & Co., 1932.

It seems a short time ago that we purchased de Kruif's "Microbe Hunters" and when we had finished reading it made the wish that without loss of time he would write another book about physicians and scientists, laboratories, test-tubes, mice, guinea-pigs, grim death and great discoveries. We doubted that he would write another book that would be as absorbing and as interesting as the one we had just finished. But in "Men against Death" the author not only has duplicated his earlier feat but many will think he has outdone it.

For here is a book that is more exciting than most best selling novels. The lay mind will be intrigued; the physician will get a thrill. In addition he will learn much. He will learn the true recital of how many discoveries were really made.

In "Men against Death" the Prologue is entitled, "How Long Can We Live." And then, in their order, we become entranced with Semmelweis, a tragic man who found a safe way to help mothers have their babies; Banting, of insulin fame; Minot, who tricked pernicious anemia; Spencer, who found an unprecedented and fantastic way to guard men from spotted fever (thrilling); Evans, who removed one great danger lurking in the American milk supply; McCoy, who fought parrot fever (a general who didn't want to die in bed); Schaudinn, who discovered the pale horror of syphilis; Bordet, who spotted the pale horror's hiding; Wagner-Jauregg, and his friendly fever; Finsen, the Dane who trapped the light of the sun; Rollier, well known for his sun therapy, especially in the treatment of tuberculosis; and Strandberg, who turned Finsen's machine-sun on tuberculosis' most desperate consequence.

A fine book. Any physician who wishes to be thrilled, entertained and receive additions to his fund of knowledge will do well to own this book.

THE CURATIVE VALUE OF LIGHT, SUNLIGHT AND SUN-LAMP IN HEALTH AND DISEASE. By Edgar Mayer, M.D., F.A.C.P., N. Y., D. Appleton & Co., 1932.

So much has been published on the subject of heliotherapy, much of it in the lay press, that it is well to have a popular book by such an authority on this subject as Dr. Edgar Mayer of Saranac Lake. The subject is discussed in a common sense fashion and the pros and cons of advanced methods of light treatment are discussed in concise yet thorough manner. While written essentially for the layman, the book will be found of interest to any medical man.

THE CARDIAC OUTPUT OF MAN IN HEALTH AND DISEASE. By Arthur Grollman, PH.D., M.D. Springfield, Ill., Charles C. Thomas, 1932.

This is a scientific monograph on the cardiac output of man which will be found of great value to the specialist and undoubtedly many physicians and surgeons will profit by a thorough study of this subject. The book is well written, easy to read and thoroughly understandable.

A THOUSAND MARRIAGES. By Robert Latou Dickinson and Laura Beam. With an Introduction by Havelock Ellis. Balt., Williams & Wilkins Co., 1932.

A Thousand Marriages was prepared under the auspices of The National Committee on Maternal Health. It is a medical study of sex adjustment; the first considerable medical analysis of marriage in its widely human relationship. In this study, we are told, the source-material came from the extensive case-histories of a distinguished gynecologist, some of which extended over a period of thirty-five or forty years. After carefully reading this work we agree with the statement that "it is by no means narrowly confined to physiological aspects. The approach was gynecological but the presentation of the whole situation, physical and psychic, is most comprehensive and searching, sane in its recognition of the facts, wise and tolerant in its attitude."

To physicians who are interested in problems of sex, whose work brings them within the borders of this subject, to gynecologists and obstetricians who feel they should go beyond the rigid borders of their specialities and iron out the sex problems of their patients, for a better and broader understanding of this subject, this book is recommended.

BOOKS RECEIVED

All books received by THE AMERICAN JOURNAL OF SURGERY are listed in this column as soon as possible after their receipt and this must be considered as adequate acknowledgement. Books that the Editor considers of special interest to our readers will be reviewed in a later issue.

ADVANCE OF MEDICINE (THE). By Lord Moynihan. Lond., Oxford Univ. Press, 1932.

ARZT UND SEINEN KRANKEN (VOM). By Albert Krecke. Munich, J. F. Lehmanns, 1932.

CHILDRENS TONSILS, IN OR OUT. By A. D. Kaiser, M.D. Phila., J. B. Lippincott, 1932.

CLINICAL GYNECOLOGY. By C. Jeff Miller, M.D. St. Louis, C. V. Mosby, 1932.

COLON, RECTUM AND ANUS (THE). By Fred W. Rankin, M.D., J. Arnold Bargen, M.D. and Louis A. Buie, M.D. Phila., W. B. Saunders, 1932.

DIAGNOSIS AND TREATMENT OF DISEASES OF THE THYROID GLAND. By George Crile, M.D. Phila., W. B. Saunders, 1932.

DISEASES OF THE NOSE, THROAT AND EAR. Ed. 3, by A. Logan Turner, M.D. N. Y., Wm. Wood, 1932.

DISEASES OF THE THYROID GLAND. By Cecil A. Joll, M.D. St. Louis, C. V. Mosby, 1932.

EARLY DIAGNOSIS OF THE ACUTE ABDOMEN (THE). By Zachary Cope, M.D. Ed. 6, London, Oxford Univ. Press, Humphrey Milford, 1932.

ERDMANN'S CLINICS. By John F. Erdmann, M.D. Phila., W. B. Saunders, 1932.

FRANKFÜRTER ZEITSCHRIFT FÜR PATHOLOGIE, Vols. I and II. Ed. by Bernhard Fischer-Wasels. Munich, Bergmann, 1932.

HANDBOOK OF EXPERIMENTAL PATHOLOGY (A). By George Wagoner, M.D., and R. Philip Custer, M.D. Springfield, Ill., Chas. C. Thomas, 1932.

INJURIES OF THE EYE. By Harry Vanderbilt Würdemann, M.D. Ed. 2, St. Louis, C. V. Mosby, 1932.

KREBSVERBREITUNG, KREBSBEKÄMPFUNG, KREBSVERHÜTUNG. By Erwin Liek. Munich, J. F. Lehmanns, 1932.

LEHRBUCH DER UROLOGIE. By Leopold Casper, Berlin, Urban and Schwarzenberg, 1932.

LET'S OPERATE. By Roy H. McKay and Norman Beasley. N. Y., Ray Long and Richard R. Smith, 1932.

MANAGEMENT OF ABDOMINAL OPERATIONS (THE). By Rodney H. Maingat, M.D. N. Y., Wm. Wood, 1932.

OXFORD MEDICAL ADVISER FOR THE HOME. By John D. Comrie. N. Y., Oxford Univ. Press, 1932.

PRACTICAL OBSTETRICS FOR STUDENTS AND PRACTITIONERS. By P. Brooke Bland, M.D. Phila., F. A. Davis, 1932.

ROUNDABOUT HARLEY STREET. By Cyril Phillips Bryan, M.D. London, John Bale Sons and Danielsson, 1932.

SYLLABUS OF LECTURES ON GYNECOLOGY FOR NURSES (A). By a Committee appointed by the American Gynecological Society. N. Y., 1932.

PERIPHERAL NERVE INJURIES

LEWIS J. POLLOCK, M.D., AND LOYAL DAVIS, M.D

TWELFTH (Concluding) INSTALLMENT

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CHAPTER XXXV

THE CRANIAL NERVES (*Continued*)

THE FACIAL NERVE (*Continued*)

The *posterior auricular* branch arises near the stylomastoid foramen. This nerve passes upward in front of the mastoid process where it is joined by a twig from the auricular branch of the vagus. It also communicates with the great auricular and small occipital nerves. As the nerve ascends between the meatus and the mastoid process it divides into two branches. The auricular branch supplies the small muscles on the cranial surface of the pinna and retrahens auriculam. The larger occipital branch passes backward along the superior curved line of the occipital bone and supplies the occipitofrontalis muscle.

The *digastric* branch has a common origin with the *stylohyoid* branch. The former supplies the posterior belly of the digastric muscle. The latter is long and slender. It passes inward to supply the stylohyoid muscle.

The *temporofacial* is the larger of the two terminal branches of the facial. It passes upward and forward through the parotid gland. It crosses the external carotid artery and passes over the neck of the condyle of the mandible. It divides into many branches which may be divided into temporal, malar and infraorbital groups. The temporal branches cross the zygoma and supply the small auricular muscles as well as the occipitofrontalis, the orbicularis palpebrarum and the corrugator supercilii muscles. The malar branches cross the malar bone and also supply the orbicularis palpebrarum. The infraorbital branches pass forward to be distributed between the lower margin of the orbit and the mouth. Some of the branches supply the superficial muscles of the face and the pyramidalis nasi muscle. Other deeper branches supply the zygomaticus, the levator labii superioris and the levator anguli oris muscles.

The *cervicofacial* division passes obliquely downward and forward through the parotid gland where it crosses the external carotid artery. Opposite the angle of the mandible it divides

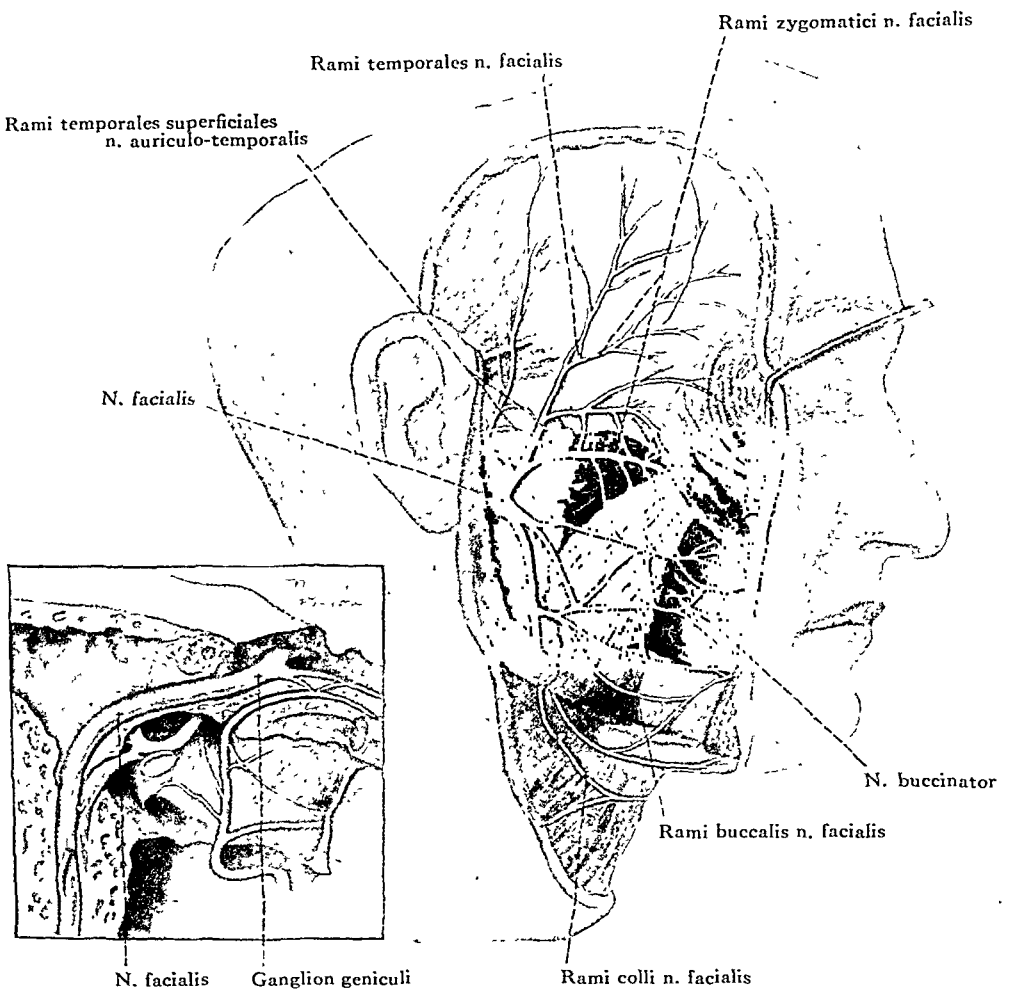


FIG. 303. Drawing to illustrate anatomical course of facial nerve.

into buccal, mandibular and cervical sets of branches. The buccal branches cross the masseter muscle and supply the buccinator and orbicularis oris muscles. The mandibular branches pass forward beneath the platysma and depressor anguli oris to supply the muscles of the lower lip and chin. The cervical branches run forward beneath the platysma

which they supply. One of these branches descends vertically to join the transverse cervical nerve.

Physiology of the Facial Nerve

As has been stated the facial is a mixed nerve. However, its major portion is motor in function. The muscles supplied by the facial are for the most part muscles of expression.

The *occipitofrontalis* muscle raises the eyebrows and the skin over the base of the nose. It also draws the scalp forward and throws the skin of the forehead into transverse wrinkles. The occipital portion of the muscle draws the scalp backward.

The *orbicularis palpebrarum* muscle is the sphincter muscle of the eyelids. The palpebral portion acts involuntarily while the orbicular portion is a voluntary muscle. When the entire muscle acts as a whole the skin of the forehead, temple and cheek is drawn inward to the inner angle of the orbit and the eyelids are closed firmly.

The *corrugator supercilii* draws the eyebrow downward and inward. The *pyramidalis nasi* draws the inner angle of the eyebrow downward.

The *levator labii superioris* muscle elevates the upper lip and carries it forward. The *levator anguli oris* raises the angle of the mouth. The *zygomaticus major* draws the angle of the mouth backward and upward while the *zygomaticus minor* draws it backward, upward and outward.

The *levator labii inferioris* raises the lower lip and protrudes it forward. At the same time it wrinkles the skin of the chin. The *depressor labii inferioris* draws the lower lip directly downward and slightly outward. The *depressor anguli oris* depresses the angle of the mouth.

The *orbicularis oris* closes the lips. It may also bring the lips in close apposition to the alveolar arch. The superficial portion of the muscle brings the lips together and protrudes them. The *buccinator* muscles contract and compress the cheeks. This action keeps the food under the pressure of the teeth during mastication. When the cheeks have been

distended with air these muscles expel it from between the lips. The *risorius* muscle retracts the angles of the mouth.

The posterior belly of the *digastric* and the *stylohyoid* muscles are important factors in the act of deglutition. After a morsel of food has passed through the pharynx the hyoid bone is carried upward and backward by these muscles. They are therefore of assistance in preventing the return of the food into the mouth.

The *sensory portion* of the facial nerve is represented by the nervus intermedius and the chorda tympani is thought to be a direct continuation of its fibers. It has been shown rather conclusively that the sensory component of the facial nerve supplies deep sensation to the face. After section of the trigeminal nerve deep pain pressure may be appreciated. In combined lesions of the fifth and seventh nerves all types of sensation are lost. Cushing and others have shown that the sensation of taste upon the anterior two-thirds of the tongue is undoubtedly carried by way of the chorda tympani and the facial nerve.

Surgery of the Facial Nerve

The facial nerve is the cranial nerve most frequently involved by trauma or disease and the one for which surgical repair is most often indicated. The nerve may be severed by a stab or gunshot wound or it may be injured inadvertently during a mastoid operation. Under such circumstances the indications for repair are quite plain. The nerve may also be torn within the cranial cavity by a fracture of the base of the skull. Cases have been recorded in which fragments of bone have compressed the nerve within the facial canal and in which simple nerve liberation have been followed by recovery. Facial paralysis may accompany an otitis media. In such cases one should wait for at least six months for spontaneous regeneration to occur. It may also follow some time after a mastoid operation in which event it may be assumed that compression and not anatomical severance has produced the paralysis.

In Bell's palsy a number of fibers to the face may function sufficiently to maintain some degree of muscle tone without a complete return of function. The nerve may be injured during childbirth by the application of forceps. Congenital maldevelopment of the facial nerve muscles may also be met but is commonly accompanied by other cranial nerve lesions.

As elsewhere in peripheral nerve surgery the ideal form of treatment is an end-to-end suture of the injured facial nerve. Because of its course within the facial canal which it fills completely this procedure is not so easy to accomplish even when the nerve has been severed by the lesion. It is therefore even more imperative that every opportunity be given for spontaneous regeneration to occur.

Occasionally the cut nerve ends may be directly sutured in an early case of division but when scar tissue and callus have developed within the facial canal this is practically impossible. Consequently other methods of nerve repair must be employed. Rarely a single autogenous graft may be used by employing a cutaneous nerve but even here the diameter of the facial canal is such that a very small amount of scar tissue may defeat an otherwise successful repair.

Frequent attempts have been made to correct a facial paralysis by the anastomosis of the distal end of the facial nerve beyond the stylomastoid foramen with the central end of another cranial nerve. The spinal accessory and the hypoglossal nerves have been most frequently used in such operations. Faure and Furet, in 1898, first performed the operation of anastomosing the spinal accessory and the facial and Körte, in 1901, first used the hypoglossal nerve in such a nerve crossing.

Many experimental investigations have been carried out to study the results of such anastomoses and many clinical cases have been reported upon. In 1900, Mannasse obtained a response in the facial musculature in three out of five dogs upon which he anastomosed the facial and spinal accessory nerves. In 1911 and later, Kennedy has reported upon similar

experimental and clinical studies. He has shown that approximately equal results may be obtained with either the hypoglossal or spinal accessory nerves. Increase of muscle tone first appeared with a definite improvement in the symmetry of the face. Return of motor function to the orbicularis oculi followed later. The eye on the affected side first closed separately but later it became associated with closure of the normal eye. He also showed that in the monkey after spinofacial anastomosis there were movements of the facial muscles associated with movements of the shoulder.

In Cushing's case of spinofacial suture there were marked movements of the facial muscles associated with turning the head or raising the shoulder. Kennedy, Grant, Hunt and others have reported marked improvement with a very small amount of associated movements. Stookey reports a case observed ten years after operation in which the only improvement was in the tone of the muscles. No emotional expression was possible. Körte stated that in his cases of hypoglossal-facial crossing voluntary movement had returned so that the orbicularis oculi contracted and the angle of the mouth could be raised.

Taylor and Clark have implanted the facial into a slit made in the hypoglossal nerve. They reported a variable return of movement in the facial musculature in all of their cases. Ballance, and Ballance and Stewart stated that the hypoglossal-facial suture was preferable to spinal accessory-facial crossing because of the fact that the cortical centers for the face and tongue are closer together. The shoulder and facial muscle movements are not synergistic and have little or no association. Frazier and Spiller also favor such an operation for the same reason. Ballance has reported a case in which the end of the facial was sutured to the hypoglossal while the end of the hypoglossal was sutured to the spinal accessory. Twenty months later this patient had a functional return in the facial muscles without associated movements of the tongue. Ballance has reported the experimental results of many investigations

in which the lingual and the descendens hypoglossi nerves have been used in such a type of operation.

The questions naturally arose during the discussion of such operations of whether or not the cortical motor center for the shoulder or tongue assumed the function of the cortical face center. Kennedy's experimental work upon nerve crossing in the extremities seemed to prove that such is the case. He found, for example, after crossing the nerve supply to the flexor and extensor muscles of the dog that stimulation of the cortical flexor center produced a response in the extensor muscles and vice versa.

Another problem always is presented in the consideration of this operation of nerve crossing. This concerns the paralysis which results from the section of the substituted nerve. Use of the hypoglossal nerve results in an atrophy of the corresponding half of the tongue. Section of the spinal accessory is of course followed by impairment of function of the arm and shoulder. The choice between these two conditions depends a great deal upon the occupation of the patient. However, as has been mentioned, attempts have been made to suture the central end of the descendens hypoglossi to the distal end of the substituted nerve with a considerable degree of success. Some feel that all things considered paralysis of half of the tongue or of the shoulder is preferable to a facial paralysis.

This has proved to be the case in a patient upon whom we operated five years ago. The patient had a complete facial paralysis on the left side which was due to a walnut-sized tumor mass between the mastoid process and the angle of the jaw. This had been present for a considerable time but her face had been paralyzed for two years. At operation the mass was circumscribed but completely surrounded the facial nerve. The mass extended into the facial canal and to remove it completely it was necessary to section the nerve high in the canal. However, a long distal segment of the facial was isolated. This was sutured to the central end of the spinal accessory. The photographs illustrate the facial appearance before, six

months, and one year after operation (Fig. 304). The return of tone in the facial muscles has been rapid so that the symmetry of her face is more nearly normal. One year after opera-



FIG. 304. Result of spinofacial anastomosis performed five years ago. A: Before operation; B: Nine months after operation; C: Four years after operation; D: Five years after operation.

tion she was unable to close her eyelid although she had good voluntary movements about her mouth. These were unasso-

ciated with movements of the shoulder. The disability produced by the impairment of function of her shoulder has not been sufficient to detract from the clinical result obtained.

Sherren collected 50 cases of facial nerve suture. A great many of the operations which had been performed were implantations or partial nerve crossings. In 8 cases the anastomosis was complete but only 2 were followed longer than six months after operation. One of these was a spinofacial and the other was a hypoglossofacial crossing. In both associated movements were present. In most of the operations, however, definite improvement in the symmetry of the face had resulted.

Schmidt anastomosed the facial and hypoglossal nerves in a patient who had been wounded by a gunshot projectile. Two and a half years after the operation the patient could draw the eyebrow inward and downward but could not contract the frontalis muscle. He could close his eye almost completely and the corner of the mouth could be drawn outward and upward.

TECHNIQUE OF SPINOFACIAL ANASTOMOSIS: The facial nerve should be exposed first. The incision should extend downward and forward from above the tip of the mastoid process. The posterior occipital vein usually crosses the line of incision and must be ligated. In making the incision through the deep fascia care should be taken not to open the parotid gland sheath. The gland should be freed carefully and held forward by a blunt retractor. The nerve may be identified usually by palpation at its exit from the stylomastoid foramen. If, however, there is a large amount of scar tissue present it may be necessary to remove the tip of the mastoid with the sternocleidomastoid muscle attachments. The digastric muscle may also make it difficult to find the nerve. While its retraction downward and backward is usually all that is necessary it may be a help to cut a part of its fibers transversely to aid in the exposure. The facial trunk should be isolated as high as possible in the canal. Sutures should be placed through the sheath just distal to the point at which it is to be sectioned.

Through the same incision the spinal accessory nerve is sought. It lies posteriorly and crosses the transverse process of the atlas before it turns downward to cross the medial surface of the sternomastoid at the junction of its middle and upper thirds. A deep layer of fascia which encloses the transverse process of the atlas and the internal jugular vein should be opened to expose the nerve. The occipital artery runs along the lower border of the digastric muscle and should be isolated and ligated.

The spinal accessory is dissected free and the point of division decided upon. Here again sutures are passed before the nerve is sectioned so as to avoid any unnecessary trauma to the nerve trunk. The end-to-end suture is then performed between the distal end of the facial and the central end of the spinal accessory in the same manner as is used in any other direct nerve suture.

Exposure of the Hypoglossal Nerve: The same incision as has been described may be used to perform a hypoglossal-facial anastomosis. However, the lower end of the incision should be carried slightly more forward. The digastric muscle is retracted upward and the sternomastoid downward and posteriorly. The carotid sheath should be opened and the hypoglossal may then be identified as it curves forward with its convexity downward to cross the internal and external carotid arteries. If there is any question about the identification the nerve may be stimulated by an electrode. The vagus, of course, has a direct course downward and lies posterior. After the nerve has been freed sufficiently sutures are placed through the sheath and it is sectioned at the point of election.

Exposure of the Descendens Hypoglossi: It is highly desirable to complete these operations by anastomosis of the distal end of either the spinal accessory or hypoglossal nerves to the central end of the descendens hypoglossi. Regeneration through such a suture line prevents sacrifice of function which follows section of the hypoglossal and spinal accessory nerve trunks.

The descendens hypoglossi runs along the lateral border of the hypoglossal nerve as it courses downward. It should be sectioned as low as possible so that its central end may be utilized for the suture without tension.

The lines of suture should be placed between or upon normal muscle tissue and protected in that manner. If the sutures are accomplished under tension a light immobilizing collar bandage of crinoline may be necessary until the line of suture is healed.

POSTOPERATIVE TREATMENT: The facial muscles should be kept from sagging and overstretching just as are the paralyzed muscles of an extremity. A simple mechanical device which will lift the angle of the mouth and the muscles of the cheek consists of an adhesive tape bridge. One end may be attached to the skin in the anterior temporal region and the other to the skin near the angle of the mouth.

We have also found Stookey's suggestion a valuable one. A small band may be worn about the head or a spring steel metal over the head like that worn by telephone operators. A strap of adhesive is attached to the skin of the face. In the free end of the adhesive a small metal hook is placed which is attached to the head band by a rubber band. This may be adjusted so that it forms an elastic rather than a fixed support. Some such a device should be worn constantly by the patient.

At the same time the patient should receive daily massage and gentle galvanic or sinusoidal stimulations to the facial muscles. Educative exercises should be taught to the patient and these should be performed before a mirror, particularly after the appearance of the first return of function.

PLASTIC OPERATIONS: The aim of plastic operations in facial paralysis is to relieve the disfiguring asymmetry of the face by elevation of the lower eyelid or the angle of the mouth. Lexer suggested the use of a pedunculated flap from the anterior border of the temporal muscle to be sutured along the lower lid to the orbicularis palpebrarum. Momburg used an aluminum bronze wire which he attached to the periosteum

of the zygoma and passed to the angle of the mouth. Kirschner, Busch, Stein and Fischer have all described and used a free transplant of fascia lata for the same purpose. Through a

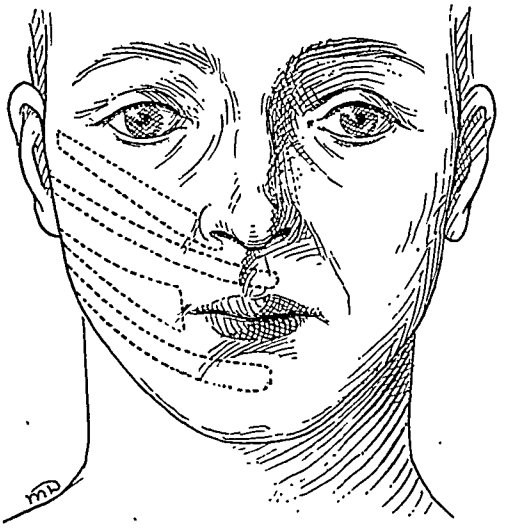


FIG. 305. Drawing of operative technique for plastic operation to relieve facial paralysis. (After Blair.)

small incision in the skin over the zygoma, the fascia lata is looped around this bone. One end of the fascial strip upon a straight long needle is pushed subcutaneously toward the angle of the mouth until it reaches a point on the upper lip 1 cm. above the commissure of the lips. The needle is pushed through the mucous membrane and the fascial strip pulled through. A small incision is made vertically downward through the mucous membrane to a point 1 cm. below the commissure. The needle is then pushed through the musculature of the cheek and is carried upward subcutaneously to the zygoma. The two ends of the fascia are pulled taut and the angle of the mouth raised as high as desired. They are then tied securely. Blair makes use of several fascia lata transplants which are anchored in the temporal fascia and are attached at the angle of the mouth, the nasolabial fold and the upper and lower lips (Fig. 305). Gersuny has joined the fibers of the paralyzed half of the orbicularis oris to those of the sound side

by making small incisions through the mucous membrane of the upper and lower lips. Failures of these operations may be traced to an insufficient amount of correction and to the fact that the grafts may shrink. All strain should be taken from the newly implanted fascia by external support of the facial tissues. Blair suggests that the mechanical fixation of facial muscles may be used in connection with a nerve crossing operation. It would limit the overstretching of paralyzed muscles and would lessen the load during the return of muscle function after such a nerve operation.

Spinal Accessory Nerve

When injured alone, the external branch which innervates the trapezius and the sternocleidomastoid muscles is involved. The internal branch, which joins the vagus soon after its exit from the cranium, supplies the muscles of the larynx and those of the soft palate. When the external branch of this nerve is paralyzed there ensues a paralysis of the sternocleidomastoid and the trapezius muscles. On turning the head to the opposite side the paralyzed sternocleidomastoid muscle does not contract and the head may be only incompletely turned to the side. In addition to the spinal accessory the trapezius muscle receives innervation from the cervical nerves so that frequently a complete section of the spinal accessory is followed by only a partial paralysis of the trapezius. The middle and infraspinous portion of the muscle is frequently spared, but when the cervical nerves are injured as well the disability is much greater. The trapezius muscle elevates the shoulder and adducts the shoulder blade toward the midline. When contracting unilaterally, the shoulder of the corresponding side is elevated, the head is drawn backward and rotated somewhat to the opposite side. When the clavicular portion of the muscle is paralyzed the shoulders do not move in respiration. When the middle portion is paralyzed the acromion falls, as the result of the weight of the arm. The upper and inner angle of the scapula is raised and displaced outward by the

levator anguli scapuli, while the inner and lower angle is approximated to the midline. The shoulder is drawn forward and downward and cannot be adducted in the usual manner.

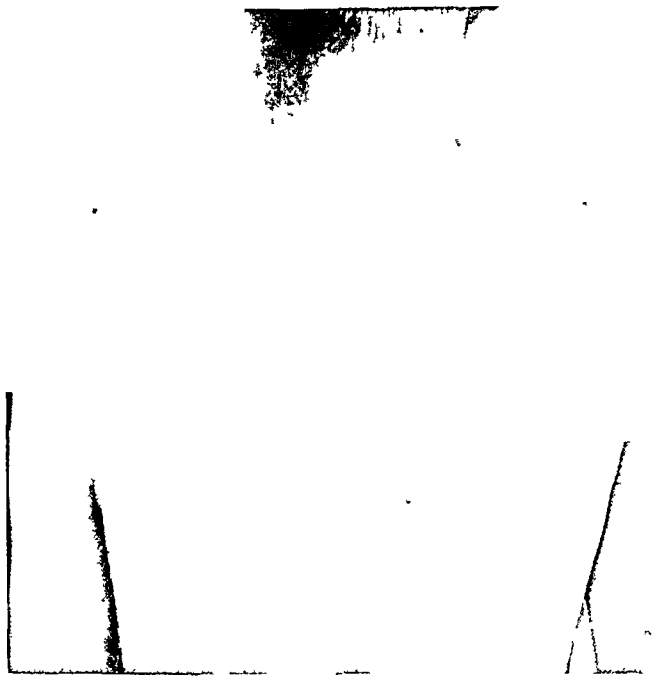


FIG. 306. Paralysis of the spinal accessory nerve.

In paralysis of the lower portion, the internal margin of the scapula is displaced outward, thus broadening the back, and the acromial portion of the clavicle is projected forward (Fig. 306).

Frequently the arm may be abducted quite adequately, but if all parts of the trapezius are paralyzed attempts to abduct the arm are followed by a forward and upward dislocation of the shoulder blade. The patient is unable to abduct the shoulder in the first third of the movement unless the shoulder blade is passively held against the chest wall.

Chapter XXXVI

EXTRACRANIAL INJURIES OF MULTIPLE CRANIAL NERVES

Simultaneous lesions of the ninth, tenth and eleventh cranial nerves have frequently been observed as the result of war injuries. Often an injury of one or more additional nerves has been present. Most frequently the twelfth, occasionally the cervical sympathetic and rarely the seventh nerve have been involved.

Extracranial lesions of the last four cranial nerves had rarely been observed prior to the war. Such cases were attributed either to compression by a tumor, gumma or tuberculous gland, or to an inflammatory condition. One of the most striking examples of such a case was reported by Beck and Hassin.

Lesions of the ninth, tenth and eleventh cranial nerves were more frequently noted as the result of non-traumatic causes about the jugular foramen. Vernet has described 6 such cases due to compression, 2 of which were observed by him. Three others were the result of inflammation and 2 of these were his own cases.

The cases which present lesions of several or all of the last four cranial nerves have been classified on the basis of symptoms added to a pure laryngeal hemiplegia. Thus, the syndrome of Avellis consists of a unilateral paralysis of the soft palate, in addition to the larynx, as the result of a lesion of the vagus and the internal branch of the spinal accessory nerves. The syndrome of Schmidt is characterized, in addition to the foregoing symptoms, by paralysis of the sternocleidomastoid and the trapezius muscles through the inclusion in the lesion of the external branch of the spinal accessory nerve. The syndrome of Jackson includes, in addition to all of these symptoms, unilateral paralysis of the tongue resulting from a lesion of the hypoglossal nerve.

Several new groups have been added by the literature of the war. Vernet has described a syndrome due to a combined lesion of the glossopharyngeal, vagus and spinal accessory



FIG. 307. Photograph of model to show extracranial course of last four cranial nerves: (—) spinal portion of spinal accessory; (=) hypoglossal; (≡) vagus; (≣) glossopharyngeal.

nerves, called by him the syndrome of the posterior lacerated foramen. Collet described a combination of symptoms due to a complete lesion of the ninth, tenth, eleventh and twelfth cranial nerves under the name of glosso-laryngo-scapulo-pharyngeal hemiplegia. The same condition was described by Vernet as the complete syndrome of the last four cranial nerves and by Sicard as the syndrome of the condyloposterior lacerated foramen. Villaret described the syndrome of the posterior retroparotid space, which is characterized by the addition of a lesion of the sympathetic nerve to the syndrome of the last four cranial nerves, producing thereby enophthalmos, narrowing of the palpebral fissure and myosis. The number of syndromes is limited only by the possible combinations

of complete or incomplete paralyses of these several cranial nerves, and the descriptive ability of the various observers.

One of the notable features of all the cases is that whatever



FIG. 308. Photograph of model to show extracranial course of last four cranial nerves: (—) spinal portion of accessory; (=) hypoglossal; (≡) vagus; (≣) glossopharyngeal.

other nerves might be affected the ninth, tenth and eleventh are rather consistently injured together. Such lesions are produced by wounds in the uppermost part of the lateropharyngeal space. This space is bounded above by the base of the skull in the region of the jugular foramen. The jugular foramen, or posterior lacerated foramen, is an opening of irregular shape and size, placed between the petrous portion of the temporal bone in front and the jugular process of the occipital bone behind. The foramen is occasionally divided into two parts by the spicules of bone which bridge it. It presents three compart-

ments. Through the anterior compartment passes the inferior petrosal sinus; through the posterior the internal jugular vein and some meningeal branches from the occipital and ascending



FIG. 309. The relation of the hypoglossal (12) and sympathetic (S) to the vagus (10).

pharyngeal arteries; and between the two veins, in order from before backward, are the glossopharyngeal, vagus and spinal accessory nerves (Figs. 307, 308, 309).

At its exit from the jugular foramen the jugular vein lies to the outside of the nerves; closely in front and internally lie

the internal carotid artery and the accompanying sympathetic nerves. At this point, Vernet states, it is possible for a projectile passing obliquely from the mastoid region on one side to the malar bone on the other to injure the three nerves and miss both the carotid and jugular vessels. This was usually the direction of the course of the missile in the cases under observation. In many cases, however, such a route was not followed. The fact that such patients survived the injury, which produced a paralysis of the three cranial nerves, without a contralateral hemiplegia, indicates one of two conditions: either severe injuries to the carotid and jugular vessels need not be fatal or result in a contralateral hemiplegia, or these cranial nerves may sustain traumatic lesions without injury of the carotid and jugular vessels. Contusion, concussion, pressure by hematomas and arterial and arteriovenous aneurysms may likewise produce loss of function of cranial nerves.

In certain respects, injury of the last four cranial nerves resembles injury of the brachial plexus. A complete brachial palsy which immediately follows injury of the plexus may improve gradually until only one or more of the cords are permanently paralyzed. In a like manner complete paralysis of the last four cranial nerves frequently eventuates in a permanent paralysis of only one, with partial lesion of one or more of the others.

In their peripheral course, the last four cranial nerves are in close proximity to a point a short distance below the level of the tip of the mastoid. Thus lesions of the ninth, tenth and eleventh cranial nerves have been produced at times by an injury not directly involving the posterior lacerated foramen.

Whether or not cranial nerves other than the ninth, tenth and eleventh are injured depends on the direction and the level of the course of the projectile. The frequent inclusion of the hypoglossal nerve in these injuries is easily understood when we recall the proximity of its exit through the anterior condyloid foramen to the jugular foramen. Likewise it is in close proximity to the ninth, tenth and eleventh cranial nerves

in the retroparotid space where its injury is frequently associated with a lesion of the sympathetic nerve. This space is described by Villaret as being bounded posteriorly by the cervical spine, internally by the pharynx, anteriorly by the internal prolongation of the parotid gland and the muscular bundle attached to the styloid process and above by the base of the skull in the region of the jugular foramen.

The symptomatology of a combined lesion of the ninth, tenth and eleventh cranial nerves is constant and easily recognized. As a characteristic triad of symptoms indicative of a complete lesion of these three nerves, Vernet proposes nasal regurgitation of fluids, dysphagia of solids and hoarseness. These symptoms represent respectively paralysis of the palate, pharynx and larynx. Which part of the collective symptomatology may be attributed to any one of these nerves is more difficult to interpret.

Considerable confusion exists concerning the innervation of the soft palate and larynx. The specific functions of the accessory portion of the spinal accessory and the vagus nerves are undetermined. Whether or not it would be more profitable to consider their functions together as those of the vagospinal nerves remains to be seen. The glossopharyngeal nerve, according to Vernet, innervates the superior constrictor of the pharynx. Loss of its function results in difficulty in swallowing solids. In lesions of this nerve, the posterior wall of the pharynx deviates to the unaffected side when the patient says "ah" with the tongue pulled forward, and there is disturbance of taste on the posterior third of the tongue. A lesion of the vagus nerve produces sensory loss on the soft palate and the posterior wall of the pharynx in addition to the well-recognized disturbances of salivation and of respiration (dyspnea or pseudo-asthma). Injury of the accessory portion of the spinal accessory nerve causes paralysis of the soft palate and larynx as well as a rapid pulse; while a lesion of the spinal portion produces paralysis of the sternocleidomastoid and trapezius muscles.

These cases are of such interest that a short description will be given of some of them.

CASE I. S. R., was wounded August 10, 1918, by a machine-gun bullet. The wound of entrance was 1½ inches to the left of the fourth cervical spine, and the wound of exit ½ inch below the anterior extremity of the left zygomatic arch. He was unconscious for half an hour. When attended by a medical officer one and one-half hours following the injury, he was hoarse, dyspneic, had difficulty in swallowing and there was marked salivation.

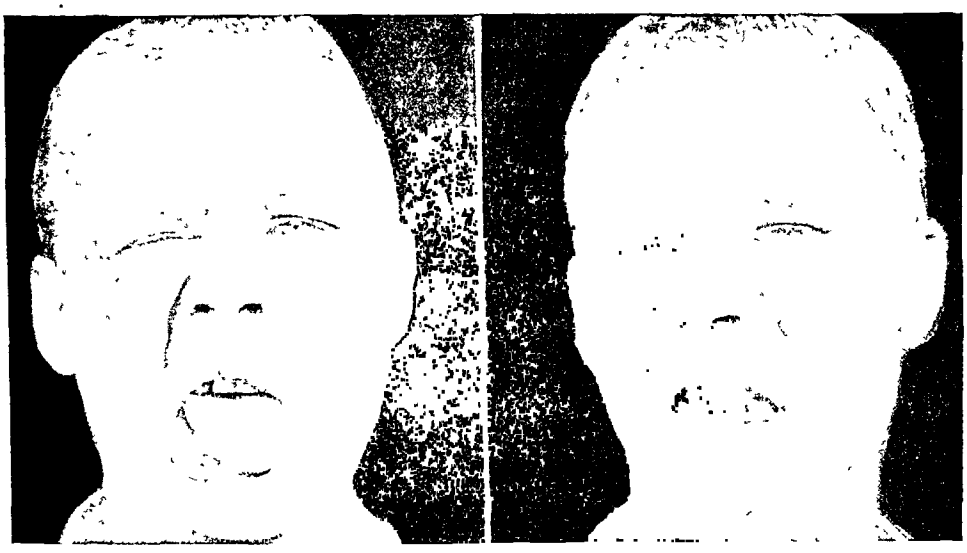
The patient was examined ten days after injury. There was a subconjunctival hemorrhage of the left eye. A hemorrhage into the mucous membrane of the posterior wall of the pharynx extended well up into the mouth of the esophagus. A peripheral paresis of the left seventh nerve and paralysis of the ninth nerve were present. He had considerable difficulty in swallowing solid food. The posterior wall of the pharynx was pulled toward the right, especially when the tongue was pulled forward. No disturbance of taste could be demonstrated.

There was paralysis of the vagospinal nerves; that is, of the accessory portion of the spinal accessory and the vagus. He had moderate salivation and sensation over the left side of the soft palate was diminished. No disturbance of respiration was noted. There was paralysis of the left side of the soft palate. The latter was pulled to the right and in phonation this deviation was increased. There was some regurgitation of fluids. The gag reflex was absent on the left. The left vocal cord was paralyzed and the voice was hoarse and nasal.

There was paralysis of the sternocleidomastoid and the trapezius muscles. On turning the head to the right only a small portion of the sternal part of the sternocleidomastoid became prominent. The shoulder drooped. The inner angle of the scapula deviated outward, the outer angle drooped and the lower angle approximated the midline and projected under the skin. The left hypoglossal nerve was paralyzed. The tongue, while in the buccal cavity, was pulled slightly toward the right. When it was protruded, it deviated to the left and showed atrophy and fibrillary twitching.

Comment: In this case we were dealing with an extracranial injury of the ninth, tenth, eleventh and twelfth cranial nerves and a partial lesion of the seventh. The course of the missile did not conform to the usual diagonal route from the mastoid region of one side to the malar of the other (Fig. 310).

CASE II. H. P., was wounded April 5, 1918, by a pistol bullet. The wound of entrance was just above the upper central incisors and the wound of exit one inch below and 2 inches behind the right mastoid process. For



A B

FIG. 310. A: Weakness of left facial and hypoglossal palsy; B: Weakness of left facial and paralysis of sternocleidomastoid.

several days following the injury he was unable to move his left arm and leg. He had difficulty in speaking, hoarseness and slight regurgitation of liquids. He preferred semisolid food, as solid food was hard to swallow.

He was examined over a year after the injury. The deep reflexes on the left side were all greater than on the right; the plantars were equal and normal; the cremasterics equal; and the left upper abdominal reflex was diminished. There were no sensory disturbances over the extremities, trunk or abdomen, and no weakness in these regions was noted. It is probable that he sustained a unilateral concussion of the spinal cord at the time of injury.

A lesion of the cervical sympathetic of the left side was shown by enophthalmos, diminution in the size of the palpebral fissure and a contracted pupil. Indicating a lesion of the ninth nerve was a history of dysphagia for solids and slight deviation of the posterior wall of the pharynx to the left on protruding the tongue. Taste was not affected.

There was a history of regurgitation of fluids and hoarseness but no history of disturbance of respiration, pulse and salivation. No objective findings were present.

In the distribution of the spinal portion of the spinal accessory there was found slight atrophy of the trapezius muscle but none of the sterno-cleidomastoid.



A B C

FIG. 311. A: Facial weakness and narrowed palpebral fissure, enophthalmos and myosis. B: Atrophy of tongue. C: Anterior dislocation of shoulder girdle on attempts to shrug shoulders.

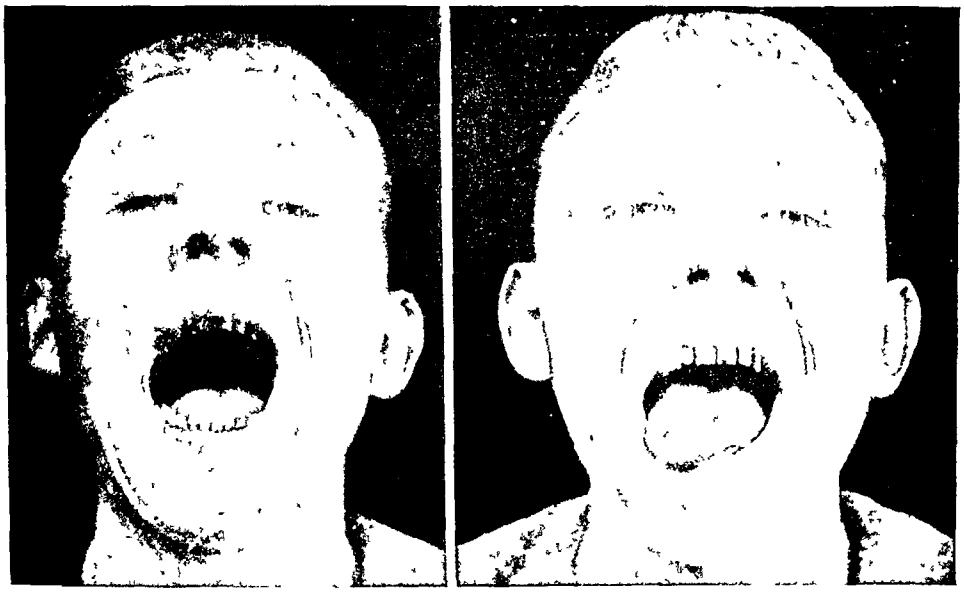
The twelfth nerve was paralyzed. The right half of the tongue, with the exception of the inner part of the very tip, was markedly atrophied. Slight paresis of the facial muscles of the right side was seen.

Comment: In this case we were dealing with a permanent paralysis of the twelfth nerve and the cervical sympathetic, and with a dissociated and recovering lesion of the ninth, tenth and eleventh cranial nerves. In addition, a slight lesion of the seventh nerve was present. This case is illustrative of the retroparotid syndrome of Villaret. Many such cases have been noted and described among others by Villaret; Faure; Lannois, Sargnon and Vernet; and Sicard and Roger (Fig. 311).

CASE III. C. L. was wounded October 10, 1918, by a high explosive shell. The wound of entrance was at the tip of the left mastoid. The missile was located by roentgenogram in front of the axis in the midline. Following the injury he was unconscious for six hours.

He was examined ten days after injury. He showed no paralysis of the extremities, trunk or abdomen. There were no sensory disturbances. No dysmetria, adiadochocinesia or other dyssynergia was found.

The findings may be divided into two groups: first, paralysis of the left facial and auditory nerves; second, paralysis of the ninth, tenth, eleventh and twelfth cranial nerves of the same side. There was a complete peripheral



A B

FIG. 312. A: Deviation to opposite side with tongue in the mouth. B: Deviation to the side of the lesion when the tongue is protruded.

facial palsy of the left side, loss of taste on the entire left side of the tongue and total deafness on the same side. He gave a history of tinnitus and in his record was a notation of gradually diminishing horizontal and lateral nystagmus to the right. Such a nystagmus of mild degree was found. There was escape of what appeared to be cerebrospinal fluid from the left ear. Because of this, caloric tests were not performed, and turning tests were omitted because of the general condition of the patient. Roentgenograms did not reveal a basilar skull fracture.

Because the peripheral facial palsy was associated with loss of taste on the anterior two-thirds of the tongue, and there was injury to both the cochlear and vestibular portions of the eighth nerve, it was concluded that the injury occurred in the peripheral part of the seventh and eighth nerves within the petrous portion of the temporal bone.

Dyspnea, hoarseness, nasal speech, palatal palsy and anesthesia and paralysis of the left vocal cord indicated paralysis of the vagospinal nerves. A complete or severe lesion of the ninth nerve was shown by paralysis of the superior constrictor of the pharynx and loss of taste on the posterior

third of the tongue. Involvement of the spinal portion of the eleventh nerve was shown by paralysis of the sternocleidomastoid and trapezius muscles. The left side of the tongue showed fibrillary twitching (Fig. 312).

Comment: The extracranial origin of the paralysis in Case III is not as certain as in the other two because of the involvement of the eighth nerve and the escape of cerebrospinal fluid from the ear. The absence of paralysis and sensory disturbances of the extremities and of cerebellar dysfunction speaks against a central origin. An intracranial but peripheral lesion would require an injury of wide extent, which the roentgenographic examination failed to reveal. The course of the missile was such that it traversed the upper part of the lateropharyngealspace. It does not seem possible that it could have avoided injury to at least some if not all of the last four cranial nerves.

The course of the missile in these cases conformed to no rule except to traverse the upper lateropharyngeal space. Whether the carotid artery or the jugular vein was injured could not be ascertained. In all of them the facial nerve was injured, as in the cases of Rimbaud and Vernet, Halphen and Bourcart, Lannois and Vernet. It is probable that many more cases of injury of the last four cranial nerves might have been recorded had they been observed soon after injury. Undoubtedly a number of them recovered sufficiently to leave a permanent paralysis of only one nerve. They were then misinterpreted when they were examined some time after injury.

Chapter XXXVII

THE RESULTS OF PERIPHERAL NERVE SURGERY

From the beginning of the late war neurologists and surgeons were surprised at the high incidence of wounds of the nerves. At first both neurologists and surgeons avoided operative interference because the results of the first operations were not those which had been anticipated from the neurological literature of civil practice. In France, M. and Mme. Dejerine advised resection of every cicatrix of a nerve trunk which manifested clinically the "syndrome of complete interruption." Their opinions influenced many surgeons, who began to perform resections and sutures.

The conditions under which many of the patients were operated upon, the pressure of the work and the lack of coordination between neurologist and surgeon, as well as the impossibility of re-examination over a long period of time, served to make the earlier statistics valueless. Only when the cases were separated into special groups and placed under the observation of trained men, and re-examination made possible, was there any opportunity for determining the value of operative procedures. This ideal condition was met with in but few instances, and most of the available statistics are based upon the observation of those other than the surgeons who operated upon the lesions. Not only is this the case, but often re-examinations could not be conducted over long periods of time by the same observer. Very often the patient could be re-examined no longer and recourse was had to questionnaires. That such a condition would lead to divergent opinions was to be expected, and the situation can well be visualized by the table illustrating the results in a group of cases of secondary suture which were collected by Foerster. (Table 1.)

The failures here may be seen to range from 3 per cent to 87.5 per cent. Of two men who had a large material, one had a

failure of 3 per cent in 370 cases, the other 64.3 per cent in 414 cases.

TABLE 1

RESULTS OF SECONDARY NERVE SUTURES (AFTER FOERSTER)

Author	Cases (Per Cent)	Success (Per Cent)	Improved (Per Cent)	Better (Per Cent)	Failure (Per Cent)
Foerster.	370	96 0	55.0	42.0	3 0
Stracker	147	75 0	13.0	62.0	25.0
Roeper	84	22.7	6.0	16.7	77.3
Ranschburg	414	35.7			64 3
Stoffel	127	62.0	23.0	36 0	38 0
Spielmeyer	100	59.0	23.0	36.0	41.0
Borchardt	59	59.3			40.7
Perthes	74	66.0			34.0
Lehmann.	69	39.1			60.9
Mauss and Kruger	42	43.0			57.0
Kunzel	44	67.7			32 3
Pelz.	18	22.2			77.8
Moro	17	82.3			17 7
Hofmann.	22	81.8			18.2
Herzog.	16	12.5			87.5
Wexberg	57	54.5			45.6
Ranzi	31	48.4			51 6
Kakula	40	70.0			30 0
Rost	24	56.5			44.5
Cassirer.	78	51.0			49 0
Steinthal	40	27.5			72 5
Total	1873	60 0			40.0

Not all of this diversity of result can be due to differences in material, facilities for good work or skill of the operators. Unless measured by similar standards such comparisons are valueless. The criteria of success differ in each instance. Foerster classed all cases as successful, which showed the recovery of a large majority of muscles so that the limb was useful regardless of whether or not sensation had returned. Under improved cases he grouped those ulnar lesions in which all but the intrinsic hand muscles had recovered, those median nerve lesions in which all muscles but the abductor pollicis brevis had regained function and those radial nerve lesions

in which all muscles but the long and short extensors of the thumb had recovered.

Many authors judged successful results by the return of function in the supinator longus in radial nerve lesions; the flexor carpi ulnaris in ulnar nerve lesions; the flexor carpi radialis in median nerve lesions; and the gastrocnemius in sciatic nerve lesions. Others speak of success judged by the ability of the patient to use the hand practically. Often a return of protopathic sensibility or shrinkage of the area of loss of pain has been used as an index of recovery. Beginning of regeneration has been used synonymously with success, and the completeness of recovery or the residuals of loss of function have often been overlooked. This is partly due to the fact that in many instances sufficient time had not elapsed following operation to permit of the evaluation of *end results.* Often when sufficient time had elapsed the patients were no longer available for examination.

In 1921, Harry Platt stated that the difficulty of keeping patients in a hospital for long periods of time was such that it explained the comparative paucity of operative statistics produced in England up to that time, and that the same deficiency was seen in the literature of the European countries. "From America," he said, "where practically every nerve injury was sustained in 1918 and the prompt segregation of all patients was affected from the beginning, and where the operations were carried out under conditions of less physical strain, valuable contributions should be forthcoming in the near future." How sadly we have failed in this respect can be seen from the total barrenness of the American literature. During 1919 the supervision of neurosurgical cases which remained under observation and which seemingly did not require operative treatment was transferred to the United States Public Health Service, under the Bureau of War Risk Insurance. In 1921, the cases were taken over by the United States Veterans Bureau. The necessity of obtaining information from three governmental sources, with different personnel,

and the discharge of most of the patients who were operated upon soon dissipated the hope of carefully following the progress of these cases.

Under the best possible conditions, reports are valuable only as they are based upon common factors in examination and the interpretation of the results of such examinations. The return of a patient to work certainly cannot be used as an index of recovery in ulnar nerve lesions, where even stenography and typing might be successfully employed. If supplementary motility is not carefully excluded many cases may be called partly successful. The early return of protopathic sensibility in certain areas, or the shrinkage of analgesia is only due to the assumption of function of adjacent uninjured nerves. Changes in the color or nutrition of the skin are valueless as indications of recovery.

The length of time following operation must be the same in comparing the results of groups of cases. In the American material sufficient time had not elapsed to permit anyone to draw any final conclusions.

TABLE II
END RESULTS OF NERVE SUTURE*

Nerve	Good	Mediocre	Negative
Brachial plexus	0	100	0
Radial	11	66	22
Median	12	56	32
Ulnar	5	72	22
Sciatic.	1	60	38
Peroneal	0	50	50
Tibial	0	50	50

* Surgeon General's Report of the Medical Department of the United States Army in the World War.

In a table in the Surgeon General's Report of the Medical Department of the United States Army in the World War, the percentage of end results of 400 cases of nerve suture is given as shown in Table II.

In addition to the statistics found in the German literature mention may be made of the following. In 158 cases of suture Forrester-Brown noted 28 per cent of complete motor recovery; 19 per cent of complete sensory recovery; 21 per cent of trophic recovery; and 50 per cent incomplete in all.

Jalcowitz reported 57 cases of resection and suture of nerves. Of these there was no improvement in 28; moderate improvement in 19; extensive improvement in 2; and recovery in none. Inasmuch as these cases were examined from seven to ten years after injury they are *end results.*

Delagénière reported 142 cases in which sutures were correctly performed. Of these there was complete success in 85.91 per cent; partial success in 11.26; and failure in only 2.81 per cent.

Wiart, in 25 cases of suture, noted 20 per cent recovery; 20 per cent improved motor function; 52 per cent failures; and .8 per cent improvement in sensation or response to electrical stimulation.

Paul Dane reported 63 cases of nerve suture. In 14, a perfect result ensued; a good result in 13; a fair one in 18; and a bad result in 18. Perfect recovery meant full motor or sensory recovery; good indicated that there was considerable recovery in motor power in some muscles; fair, that there were some signs of recovery, such as diminution in the area of anesthesia or change in the tone of the muscle. Kennedy reported that 73 per cent of all sutures were completely successful.

Stopford described the results of secondary suture of peripheral nerves accurately and was able to observe his cases for a long time and make many examinations. His conclusions are among the most important available. He reported upon 271 cases. From the tables illustrating his article we have made the following analysis. Of 245 cases, there were 30 failures and 16 had shown no signs of regeneration in from six to thirteen months. A number of cases in which protopathic sensibility had begun to return were included among the successes.

Platt, in an equally accurate analysis, reported 150 cases of sutures with recoveries in 79 per cent and failure in 21.3 per cent. Sensory recovery alone was noted in 16 cases.

Our own material[1] is practically valueless from the standpoint of end results of secondary sutures. Of 81 cases, only 42 were examined at intervals more than three months after suture. Of these 27, or 64.28 per cent, showed some recovery in motion or motion and sensation; in 3 sensory regeneration, *not overlap*, had occurred alone.

Interval between Operation and Injury

It is generally accepted that the influence of the time factor has been definitely proved. Little difference is seen in the cases operated upon within eighteen months following injury, although Stracker has pointed out that the quality and type of regeneration is definitely affected.

Ranschburg had 59 cases operated upon less than six months after injury, with 47 per cent successful results; 147 cases operated upon six to twelve months, 41 per cent; one to two years, 25 per cent; more than two years, 9 per cent.

Of 21 cases operated upon within three months after injury Stracker reported success in 66 per cent; four to six months, 46 cases, 50 per cent; seven to twenty-two months, 69 cases, 24 per cent.

Stopford believed that a delay of from twelve to eighteen months appears to have no marked effect upon the date or extent of recovery. If the interval exceeds this time, the prognosis is not so good when the suture has been performed in the distal part of the limb; whereas in the proximal part a delay of two to three years does not seem to prejudice the chances of success.

Foerster's view as to the influence of time upon the beginning of recovery may be seen from the following table:

[1] Almost all of these cases were operated upon by Dr. Dean Lewis.

TABLE III

INFLUENCE OF TIME INTERVAL UPON BEGINNING OF RECOVERY (FOERSTER)

Interval between Wound and Operation Months	Number of Cases	Complete Section Recovery began Months
1	3	4.6
2	24	5.8
3	36	3.8
4	48	5.2
5	61	4.8
6	37	5.1
7	26	5.0
8	17	5.2
9	24	5.9
10	24	5.5
11	12	4.4
12	20	5.2
13	9	
14	2	
15	3	
16	3	
17	4	4.1
18	1	
19	1	
25	1	
29	2	

Jalcowitz also classified his cases with reference to the results, together with a consideration of the interval elapsing between injury and operation (Table IV).

TABLE IV

INFLUENCE OF TIME INTERVAL UPON BEGINNING OF RECOVERY (JALCOWITZ)

Interval (Months)		Improvement			Recovery
		None	Moderate	Extensive	
Less than 3	11	4	6	1	
3–6	27	7	15	5	
6–9	31	13	15	3	
9–12	15	12	3		
More than 12	12	10	2		
	96	46	41	9	

Dane tabulates the time interval as follows:

TABLE V

INFLUENCE OF TIME INTERVAL UPON BEGINNING OF RECOVERY (DANE)

Result	Interval of Time in Months between Wound and Operation
Ulnar Nerve	
Perfect	9, 1, 10, 3, 4
Good	9, 15, 5, 3, 12, 14, (1 unknown)
Fair	1, 1 day, 8, 9, 4
Bad	9, 7, 14, 7, 10, 1 day
Median Nerve	
Perfect	7, 9, 5
Good	10
Fair	1 day, 7, 3, 4, 14, 3, 8, 12
Bad	15, 5
Sciatic Nerve	
Perfect	7, 2 weeks, 4
Good	6, 12, 18
Fair	10
Bad	1 day, 7, 7, 34, 25, 12
Musculospiral Nerve	
Perfect	3, 6, 2
Good	11, 12
Fair	14, 9, 15, 7
Bad	13, 11, 10

It has seemed to many authors (Athanasio-Bénisty, Mendelssohn, Braquehage and Carriere) that primary sutures result in relatively rapid and constantly favorable results. Of 9 such cases in our series all showed complete motor recovery; 6 complete sensory recovery; and 3 partial sensory recovery, *not overlap.*

INDIVIDUAL NERVES

The statistics relative to individual nerves are exposed to the same inaccuracies noted above and, in addition, it is necessary to emphasize that in ulnar nerve lesions, supplementary motility is so extensive that many errors are possible in interpreting possible regeneration. This is equally true of combined ulnar and median nerve lesions where the strength of movement of the phalanges does not indicate which nerve

TABLE VI

RESULTS OF NERVE SUTURES UPON INDIVIDUAL NERVES (AFTER FOERSTER)

Nerve	Foerster				Stracker			Ranschburg			Spielmeyer				Stoffel			Lehmann			Borchardt			Perthes			Summary		
	No.	S %	I %	F %	No.	S %	F %	No.	S %	F %	No.	S %	I %	F %	No.	S %	F %	No.	S %	F %	No.	S %	F %	No.	S %	F %	No.	S %	F %
Radial	109	56.0	39.4	4.6	43	74 0	26.0	121	52.0	48.0	32	34.3	34 3	31 3	22	68.2	31.8	18	61.0	39 0	20	75.0	25.0	32	72.0	28.0	397	72.0	28.0
Median	83	41.0	56.6	2.4	23	83.0	17.0	47	27.5	27.5	16	25 0	25.0	50 0	4	50.0	50.0	11	36.4	63.6	3	66.6	33.4	11	73.0	27.0	198	69.0	31.0
Ulnar	64	43.9	54.9	0.8	40	57 0	43.0	72	33.0	67.0	12	25.0	8 4	66.6	5	40 0	60.0	15	26.7	33.3	9	33 4	66.6	11	64.0	36.0	228	57.0	43.0
Musculocutaneous	22	86.4	13.6	0.0	..	...		11	36 4	63.6	..		...	.	..	...	...	.			..	...	...	..	.		33	82.0	18.0
Axillary	16	81.2	8.8	0.0	..		...	4	25.0	75.0	..			.	..	...	...	.			..		.	..	...		18	78.6	21.4
Suprascapular	8	87.5	12.5	0.0																									
Thoracodorsalis	8	100.0	00.0	0.0																									
Subscapular	8	100.0	00.0	0.0																									
Long thoracic	6	50.0	50 0	0 0																									
Sciatic	44	34.0	60 0	6.0	17	100.0	00.0				21	4.8	53.4	42.8	5	20 0	40.0	18	38.9	61.1	10	20 0	80 0	6	83 0	17.0	121	70.0	30.0
Peroneal	16	62.5	31.3	6.2	20	80 0	20.0	89	16.9	83.1	9	22.2	44.4	33.4	5	60.0	80 0	6	00.0	100 0	..		...	7	57 0	43.0	152	38.0	62.0
Tibialis	10	60 0	40.0	0.0	4	75.0	25.0	38	39.5	60.5	2	00.0	100.0	00 0	..			1	100.0	00 0	..	...	...	2	00 0	100.0	67	46.0	54.0
Femoral	2	00.0	100.0	0.0																									
Inf. gluteal	1	100.0	00.0	0.0																									

No. = Number of cases
S = Success
I = Improved
F = Failures

is regenerating. The very large area of overlap of protopathic sensibility in median nerve lesions makes it possible to misinterpret this for regeneration.

Table VI indicates the reported results in the various nerves.

In addition to this, Tables VII to XII may be added:

TABLE VII

RESULTS OF NERVE SUTURES UPON INDIVIDUAL NERVES (DANE)

Nerve	Perfect	Good	Fair	Bad	Total
Ulnar..	5	7	5	6	23
Median...	3	1	8	2	14
Radial....	3	2	4	3	12
Sciatic..	3	3	1	6	13
Musculocutaneous.	0	0	0	1	1

TABLE VIII

RESULTS OF NERVE SUTURES UPON INDIVIDUAL NERVES (DELAGÉNIÈRE)

Nerve	Number of Cases	Complete Success	Partial Success	Failures
Radial	47	37 (82.9%)	7 (14.89%)	3 (6.34%)
Ulnar	23	19 (82.6%)	3 (13.04%)	1 (4.34%)
Median	15	13 (86.66%)	2 (13.33%)	0
Musculocutaneous	5	5 (100%)	0	0
Brachial plexus	2	1 (50%)	1 (50%)	0
Sciatic nerve trunk	25	23 (92%)	2 (8%)	0
Peroneal	15	14 (93.33%)	1 (6.66%)	0
Tibial	4	4 (100%)	0	0
Posterior tibial	6	6 (100%)	0	0
Total	142	122 (85.91%)	16 (11.26%)	4 (2.81%)

TABLE IX

RESULTS OF NERVE SUTURES UPON INDIVIDUAL NERVES (FORRESTER-BROWN)

Median (35) 50% comp. motor; 28% comp. sensory
Ulnar (53) 17% comp. motor; 13% comp. sensory
66% incomplete of both
Radial (21) 62% comp. motor; 33% comp. sensory

TABLE X

RESULTS OF NERVE SUTURES UPON INDIVIDUAL NERVES (PLATT)

	Total	Proximal Muscles Only	Proximal and Distal (Incomplete)	Proximal and Distal (Total)	Distal Alone	Associated Sensory Recovery	Sensory Recovery Alone	Number of Recoveries	Failures
Radial.....	35	10	11	5		all		26	9
Median—									
Upper arm...	10	7	3			in 5		10	nil
Forearm.....	20	3			6	in 6	8	17	3
Ulnar—									
Upper arm...	27	19	3		1	in 14	1	24	3
Forearm.....	20				10	in 14	7	17	3
Sciatic—									
Trunk.....	21	16	nil	nil		in all		16	5
Segmental....	4	2	nil	nil		in all		2	2
Peroneal.....	9	4	nil			in all		4	5
Posterior interosseus.....	1							nil	1
Plexus.....	3	2				2		2	1

Recoveries 118 = 79 per cent
Failures 32 = 21.3 per cent

TABLE XI

RESULTS OF NERVE SUTURES UPON INDIVIDUAL NERVES (STOPFORD)

Nerve	Cases	Failures	No Evidence of Recovery after Six Months
Radial.....	58	8	
Median.....	96	7	9
Ulnar.....	36	8	2
Sciatic.....	31	3	2
Peroneal.....	22	5	4
Tibial.....	2	0	0

TABLE XII
RESULTS OF NERVE SUTURES UPON INDIVIDUAL NERVES (POLLOCK-DAVIS)

Nerve	No Recovery after Three Months	Beginning Recovery after Three Months
Median	6	4
Ulnar	6	3
Peroneal	7	7
Sciatic	5	2
Radial	11	10
Ulnar and median	7	5

LEVEL OF THE LESION

The distance of the injury from the distal extremity of the limb seems to be an important factor in determining the time of recovery. Foerster makes the following notes relative to this:

	Months
Median—ulnar: lesions in axilla	12
lower third forearm	3
Sciatic: in gluteal region	9–11
just above knee joint	4– 6
Radial: in plexus—sup. longus recovery	12.4
level of lat. dorsi insertion	8
lower third arm	3

Platt notes that after suture of the radial nerve in the upper, middle and lower third of the arm return of power may be manifested in the supinator longus at about the seventh, sixth and fourth month respectively. Stracker was unable to find recovery in the distal muscles supplied by the radial, median or ulnar nerves irrespective of the level of the suture before one year had elapsed. He felt that in general sutures below the origin of the proximal muscle branches gave bad results. Platt believes that if the results of suture of the median and ulnar nerves in the upper arm or forearm be compared as regards distal recovery alone the quantitative recovery appears to be roughly equal in each case. Stopford is of the opinion that a nerve fiber regenerates more rapidly

TABLE XIII

RATE OF RECOVERY OF INDIVIDUAL MUSCLES (FOERSTER)

(Radial Nerve Lesion in the Axilla)

	Months
Long head triceps.	3
Lower median head triceps	4
Supinator longus	6
Extensor carpi rad. long	8
Supinator brevis	13
Extensor carpi rad. brev	13
Extensor digiti comm	14
Abductor pollicis longus	15
Extensor carpi ulnaris	16
Extensor pollicis longus	17
Extensor pollicis brevis	19
Extensor indicus proprius	19

(Median Nerve Lesion in the Middle of the Arm)

	Months
Pronator teres	4
Palmaris longus	6
Flexor carpi rad	10
Flexor digiti sub	6
Flexor digiti prop	12
Flexor poll. long	12
Opponens long .	18
Flexor poll. brevis	18
Abductor poll. brevis	22

(Ulnar Nerve Lesion in the Lower Third of the Arm)

	Months
Flexor carpi ulnaris	3
Flexor digiti prop	5
Adductor pollicis	13
Interossei	16

(Peroneal Nerve Lesion about the Knee Joint)

	Months
Peroneus longus	6
Peroneus brevis	9
Extensor dig. longus	9
Tibialis anticus	13
Extensor hallucis	19

(Tibial Nerve Lesion about the Knee Joint)

	Months
Gastrocnemius	5
Soleus	5
Tibialis posticus	8
Flexor digit. longus	15
Flexor hallucis longus.	15
Plantar muscles	24

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and readily the nearer the suture has been made to its anterior corneal cells. Although the example above of the spinator longus does not support this, some other muscles in his records of radial and median nerves do.

As has already been observed under the individual nerves, the order of recovery of muscles in injuries of the various nerves is reported in a varying manner by different observers. Foerster's observations upon his large number of cases are exceedingly valuable in this respect and the conclusions are noted in Table XIII.

Time of Recovery

Reports of immediate and very early regeneration may be interpreted as due to errors of interpretation of supplementary motility and sensory overlap. Foerster noted the following time for the various nerves observed by him:

Table XIV

TIME OF BEGINNING OF RECOVERY AFTER NERVE SUTURE (FOERSTER)

Nerve	Complete Section	
	Beginning of Recovery (Months)	Latest Beginning of Recovery (Months)
Radial	4.2	11
Median	3.2	9
Ulnar	3.5	9
Musculocutaneous	3.8	7
Axillary	4.0	9
Peroneal	6.5	12
Tibial	4.1	8
Sciatic	7.0	16

End Results

The final result may fall short of perfect recovery although regeneration may have started and progressed satisfactorily for a time. In lesions of the radial nerve, although all of the muscles may have recovered function, individual movements of extension of the proximal phalanges may remain faulty.

The small hand muscles in ulnar and median nerve lesions are often imperfectly recovered and the movements remain clumsy and inaccurate. At times this is due to imperfect sensory regeneration, especially of the fibers which carry deep sensibility.

Neurolysis

A considerable number of neurolyses and hersage have been performed. There is some difference of opinion as to the indication for this surgical procedure. Although recovery occurred in a relatively large percentage of cases, the question arises as to whether it would have occurred had the neurolysis not been performed. Athanasio-Bénisty reports upon 50 "simple freeings," performed by Gosset and Pascolis, with 20 complete cures and only one failure. Hersage performed upon 50 to 60 cases resulted in 25 recoveries and only one failure. She says, however, "It is a question whether operations of this kind are essential. Thus, an injured nerve which has not suffered severe cicatricial damage and has maintained its continuity will probably be able to regenerate at the end of eight to twelve months without an operation of any kind." In regard to the time element of this question, it may be stated definitely that although it was found that severely injured nerves began their recovery, and regeneration proceeded much as if the nerve had been sutured, a large number showed little regeneration twelve months following injury. Platt likewise believed that although in his 80 cases there was improvement in 75 per cent, it has been impossible to prove that the operation alone has determined this.

Foerster performed neurolysis 194 times and followed 188 of his cases. He reports a successful recovery in 77.2 per cent; 2.7 improved and 21 per cent failures.

In our small material of 39 cases of neurolysis, 35 were examined after two months. Of these, 27, or 73 per cent, showed continued regeneration.

TABLE XV
RESULTS OF NEUROLYSIS (AFTER FOERSTER)

	Cases	Successful Results (Per Cent)
Mauss and Kruger	35	80.0
Kunzel	23	65.2
Stoffel	48	68.7
Pelz.	36	55.5
Moro	11	100.0
Stracker	91	83.5
Borchardt	30	80.0
Wexberg	59	81.3
Ranzi	33	76.0
Ranschburg	405	37.2
Perthes	49	69.4
Lehmann	46	54.5

NERVE TRANSPLANTS

In general, transplants were not followed by the degree of regeneration which we were led to believe would occur as the result of animal experimentation. In our own material regeneration was seen to have begun in 2 of 7 cases of radial nerve injury. The cases of injury to other nerves were observed too short a time after operation to permit of any conclusion.

TABLE XVI
RESULTS OF AUTOTRANSPLANTS (FOERSTER)
(21 Cases)

Radial Nerve (5)	1 success (25 months); 1 improved (8 months) 2 improved (11 months); 1 failure (10 months)
Median Nerve (4)	1 success (14 months) 1 improved (12 months)
Ulnar Nerve (11)	2 success (5–6 months) 8 improved (2 months)
Axillary Nerve (1)	success (28 months)

The results of homoplastic and heterotransplants in Foerster's hands were all bad. Gosset and Chairier analyzed

216 cases of nerve grafts. Only 5 good results were seen in the autografts; 2 in homografts; and 5 in heterografts.

A few successes were reported by Senient, Joyce, Forrester-Brown and others. Platt reported 18 "bridge operations" with complete failures. He had occasion to re-operate upon 6 cases, and Stopford examined 7 additional cases, all of which had shown failure of recovery. Platt was unable to confirm the presence of hypertrophy of the grafts described by various observers (Joyce, Lewis, Forrester-Brown). He unhesitatingly condemns the use of nerve grafts. It is to be noted that in England the use of single cutaneous grafts, and not cable transplants, had been the practice. We must await further observations on a large material of cable transplants of large size to confirm the results of a few observers before any final conclusion can be reached as to the efficacy of this operation.

[*The End*]

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